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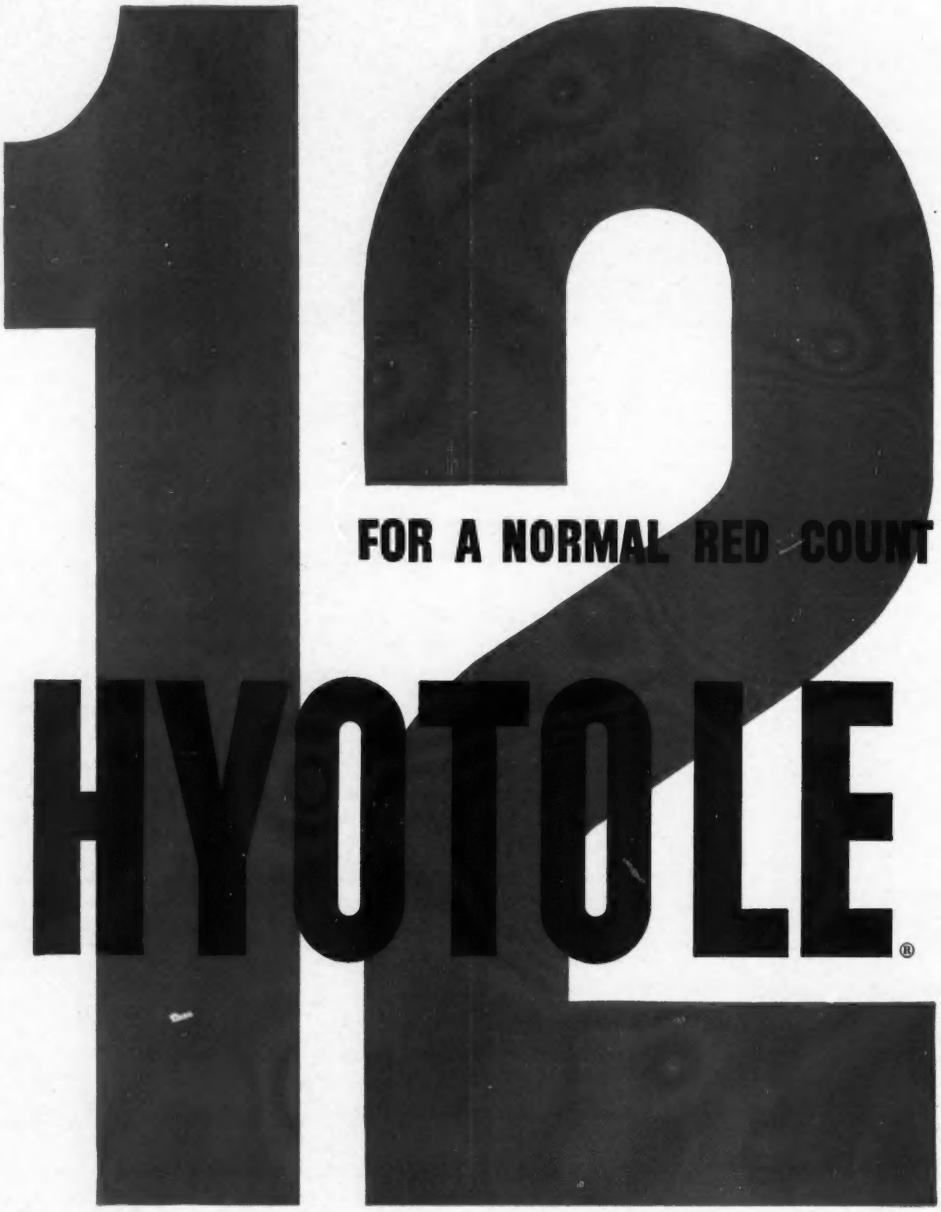

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The Meaning of Disease

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• A wider concept of disease is developing, which deals with the social environment, not only with the physical, chemical or ecological factors, as they affect the homeostasis of the internal environment of the organism. In such a concept it is the fitness of the whole personality which determines ease or dis-ease in adaptation. If the medical profession is to retain the strategic direction as well as the tactical command of the battle for health, it must widen the bases of its educational program so that every physician will understand and conform to the plan of battle even though his individual role is highly restricted; so that even in the office of a technical specialist the whole personality of the patient in relation to his whole environment is dealt with.

ABOUT 25 YEARS AGO there appeared a remarkable and rather epoch-making book by William Alanson White under the title of *The Meaning of Disease*,[2] in which he developed the then rather revolutionary concept that disease was a manifestation of man's struggle to adapt to his environment. Until then medical thought about disease had been preoccupied with the pathological processes affecting the structure and functions of organs rather than with the struggle taking place between the owner of those organs and the total environmental situation in which he exists.

In the *Bible*, of course, were many references to the relationship between the perturbations of the soul and those of the body, but, in the main, religion kept the concepts of the two in quite separate compartments. Burton in his *Anatomy of Melancholy* attempted to deal with this relationship on a philosophical basis. Claude Bernard had taught that the organic functions of the body served to keep its internal environment within a relatively constant range of conditions. It remained for Cannon to show experimentally that these physiological mechanisms were profoundly affected under the stress of emotional reactions. Thus psychosomatic medicine was born legitimately of Psyche and Soma, and christened at the font of science. It was White who made us see that these psychosomatic disorders did not occur piecemeal, organ by organ, but that all illness or disease must be conceived as the struggle of the *whole* personality against the stresses of the *whole* environment.

This idea has today achieved the widest acceptance, as one may see in reading the symposium on "Adaptation" edited by John Romano,[1] in which a general biologist, a social anthropologist, a psychologist, a physiologist, and a psychiatrist met on common ground. In spite of this agreement in the upper echelons of Olympus, the rank and file of mortal practitioners are still committed to the old concepts

Address of Guest Speaker, presented before First General Meeting at the 81st Annual Session of the California Medical Association, Los Angeles, April 27-30, 1952.

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of diseases of the parts of man and with parts of the environment rather than with the struggle of the whole man with his whole environment. Thus it seems pertinent to discuss the *meaning* which disease must have to each physician both as an individual and as a member of a great profession. If we grasp this meaning, if we accept the wholeness of things pertaining to disease, how can we continue to categorize diseases according to the deficiencies of organic responses? Can we persist in the narrow sort of specialization in practice which has been engendered by the older concepts? Can we go on treating only the eye, the ear, the heart, the lung, the stomach, bowel, or prostate of a patient without consideration of the whole man and the setting in which he exists? Are we, like Joseph's brothers, so busy dividing up Joseph's coat of many colors that we forget what becomes of Joseph?

"Well," you will say, "the capacities of even the greatest doctors are finite, while the problems of medicine are infinite. Let each man play the part for which he is best fitted—if worse comes to worst, we have *groups*. At any rate that is what is happening, so make the best of it."

What about *group practice*, is it to be a congeries of technical experts who pass a patient down a disassembly line and back up an assembly line? Is there anyone to comfort the sick man and hold his hand, and be guide, philosopher and friend, while he goes through this process? What about the *group brain* and the *group conscience?* Is the group going to confine itself only to patching up the man's body or is it going to help him to achieve a better total adjustment to his environment, or to seek a new one for which he is better fitted to adapt? These are pertinent questions to which the answers are being hammered out every day on the anvil of trial and error.

Let it not be assumed, from the questions I have asked, that I am against groups in medicine. Nothing becomes more apparent, as our understanding of disease progresses, than that the care of the sick is becoming a team operation, the success of which depends on a captain, imbued with the highest ideals of sportsmanship, with a good eye to strategy as well as to the tactical formations which respond to the signals which he calls. He and his whole team must have the broad concept of the nature of disease, and realize that each patient is struggling with his entire social and ecological environment and not simply with trauma, infection, intoxication, organic defect, or other immediate cause of an episode of illness. At best the medical group operating a clinic represents a team dealing with the immediate problems of illness, chiefly those of diagnosis and therapeutics.

A good group will take such immediate measures as may be available to *prevent* recurrence or extension of an illness. It may employ the ancillary services of visiting nurses and social workers to meet environmental problems of the home and family, and to effect liaison with public agencies which are a part of the forces of preventive medicine.

A good group may also organize facilities to aid in rehabilitation, although the requirements for the conduct of this "third phase" of medicine will be far beyond the capacities of most groups, just as are those of preventive medicine.

If one estimates the situation confronting medicine in the Battle for Health, one can readily see that three types of teams are required: one for *diagnosis* and *therapeutics*, one for *prevention*, and one *for rehabilitation*.

Our traditional concepts of the functions of these forces are quite inadequate in the light of what we may presently conceive to be the nature of disease. We have a better understanding of the requirements of a diagnostic-therapeutic group than of the table of organization for the preventive or rehabilitative forces. We have much to learn about how to interrelate them and coordinate their tactical activities, and the grand strategic plan has yet to be drawn.

In the field of preventive medicine, it is obvious that great strides have been made in reducing the bill of mortality by the efforts of our public health agencies directed against the immediate microbiological causes of disease. It cannot be said, however, that we have yet achieved much in the reduction of total morbidity. In fact the reduction in the death rate in the earlier years of life has probably even increased the amount of chronic illness, for more have been spared to be its subjects. The forces of prevention are being directed with great energy against the immediate or proximate causes of morbidity in industry and in senescence, and against cancer and infection. In the social sciences and in psychology the primitive concepts of social pathology and psychopathology are forming. The psychiatry to which we must look for guidance in this phase of the battle is rapidly approaching the point where it may be able to assume the leadership which medicine must give in the fields of child training, education, both secular and religious, and in the fields of law and public administration.

What is the kernel of the problem that confronts us? Is it not that the growth of science and technologic development have enormously extended the scope of environmental stresses to which man must adapt? This expansion in scope has been so rapid that the older mechanisms which society has employed for bringing up a majority of its members from the infantile state of primitive little beasts to full stature as mature adult men and women, have proved inadequate, in the sense that only a small

proportion achieve maturity and a larger proportion remain immature. Within the short space of 300 years we have gone from societies in which a majority of citizens might live out their span of years within the confines of a single county to one which is truly global in extent, owing to developments in communication and transportation. In smaller and more circumscribed societies when the rate of change was not too great, a semi-static equilibrium tended to develop. There was some sort of harmony attained by religious and secular education which developed a sufficient body of mature adults to manage community affairs successfully. In our greatly expanded society we need a mature psychiatry to guide us in child training, in improving the methods of bringing children to the maturity demanded by our global situation. Our mores and our laws, our ethical goals must be set to a realistic conception of our situation and more people must be brought to mature adaptation to it. This is the final task of preventive medicine.

The concepts of rehabilitation have developed from our better understanding of the meaning of disease. In the normal development of the mature person there is an orderly *progression* through stages of infantilism, childhood, early and late adolescence to adulthood. We recognize that *regression* takes place in illness, by which we mean that the personality reverts toward earlier levels of maturity when one is ill. For instance, the man who is so ill that he must be fed and bathed and have a bed pan and urinal brought to him, returns in a sense to an infantile level, from which he must again progress as convalescence takes place. It is the function of rehabilitation to promote this reprogression. There is far more to this than the restoration of function to an injured part, more than the fitting of prosthetic appliances. All the resources for psychological rehabilitation must be employed as well as those of physical medicine.

The processes which make for successful convalescence offer medicine an unrivalled opportunity to carry the process beyond the level preceding illness. One could mention innumerable instances in which the conquest of severe disability has carried an individual to new heights of maturity and achievement, far beyond those which were in evidence before illness or disability occurred.

For guidance in this "third phase" of medicine we must expect leadership from psychiatry. This field of medical endeavor is rapidly emerging from the state in which it was regarded as a specialty, devoted to the treatment of psychoses. Psychiatry is no longer immured within the walls of madhouses; it is taking its place in the wards of general hospitals, and in health departments. In these roles it is developing and extending its scope so that it is no longer a specialty devoted to the care of the insane and to the lesser forms of neurotic illness. Psychiatry will become one of the most basic parts of the training of the general practitioner and the internist, who is, after all, a general practitioner with a big reputation.

In the medical school in which I serve, our department of psychiatry has teachers assigned to each of our other major clinical services. It is the function of those teachers to study, analyze and interpret the psychological phenomena attending all types of illness. The clinical clerk who is assigned to a patient with peptic ulcer will be able to synthesize the lessons learned with the psychiatrist as well as with the gastroenterologist and the surgeon. These latter gentlemen are also being instructed and all of us are functioning better as a team because of this leaven in our daily bread. It is a common experience for me on my ward rounds to have a student tell me that a certain patient with hypertension is unable to express anger overtly, or that Mrs. Jones, who has ulcerative colitis and was doing well, has relapsed because the doctor on whom she was dependent has left town for a vacation; or that Mrs. Brown, who last week was in status asthmaticus, has remarkably recovered on the news of the death of her husband.

We oldsters have learned the truth of many of these things the hard way, and slowly, over many years of experience. These youngsters on completion of their training will approach their various tasks with a breadth of view and with a perspective that was denied us in our own school days. We can look forward then to a time when we will possess a corps of physicians who can assume strategic direction as well as tactical command of the three great phases of the battle for health, so that each will understand and conform to the plan of battle, which deals with the whole personality of the patient in relation to his whole environmental situation, even though the individual physician's role in the team is highly specialized.

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The Clinical Picture of Pancreatic Insufficiency

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• Minor degrees of pancreatic insufficiency may go unrecognized. There is a paucity of symptoms and physical findings in mild and moderate degrees of insufficiency and in such circumstances laboratory methods are necessary to determine the presence of insufficiency. The clinical picture when insufficiency is well established may be characterized by loss in weight; vague indigestion; voluminous, light-colored, glistening stools in which fat globulets may be seen; changes in the concentration of pancreatic enzymes in the blood indicative of lowered pancreatic function; diminished amounts of pancreatic enzymes in the duodenal juice, and the related poor digestion of fat and protein in the food. Lowered tolerance of carbohydrate, as found in diabetes mellitus, may or may not be present. The location and character of the disease in the pancreas causing the insufficiency may or may not be apparent.

THERE ARE TWO KNOWN FUNCTIONS of the pancreas, that of internal secretion of insulin, having to do with the metabolism of carbohydrate, and that of external secretion of enzymes, important in the process of digestion. The insufficiency of insulin results in the classic disease of diabetes mellitus. The insufficiency of the external secretion may be more obscure in its clinical manifestations. Pancreatic insufficiency, with or without disturbance in carbohydrate metabolism, may be related to any of the diseases of the pancreas which cause destruction or impairment of function of the acinous glandular tissue, such as chronic pancreatitis, tumor, hemorrhage, or stones.

Pancreatic insufficiency, theoretically, may arise to some degree in other conditions in which the nervous and/or humoral mechanisms of pancreatic secretion are disturbed, but probably so-called functional pancreatic insufficiency is of little or no clinical significance, since it would be expected to be so mild, transient and masked by other symptoms as to be difficult of recognition.

The external secretion of the pancreas contains three important enzymes: amylase, which acts on starch; lipase, which splits fats; and trypsinogen, which, after activation to trypsin, acts on proteins. Insufficient amounts of these enzymes reaching the intestinal tract produce the clinical features of the inadequate digestion of these three elementary food materials. Minor degrees of insfficiency go unrecognized. The digestion of fat and protein is not greatly impaired until there is a pronounced decrease, probably up to 75 per cent, in the lipase and trypsinogen below normal; and an even greater diminution of amylase is necessary before carbohydrate digestion is measurably impaired. The appearance of gross evidence of the indigestion of these food elements, of which fat indigestion is the most easily recognized, varies with the nature of the disease causing the insufficiency. In chronic pancreatitis the disease process, except for the acute episodes, is slowly progressive, and the changes resulting from the indigestion may not appear clinically for weeks, months or years after the onset of the disease. Evidences of indigestion are immediate if complete obstruction of the pancreatic duct occurs. Partial obstruction of the pancreatic duct may interfere with the egress of the pancreatic juice only enough to make indigestion slower or intermittent in appearance.

Minor degrees of pancreatic insufficiency may remain obscure or go unrecognized in chronic alcoholism, Laennec's cirrhosis of the liver, and gallbladder disease.

Diseases which may have an associated pancreatic insufficiency are acute pancreatitis, carcinoma of the pancreas, carcinoma of the ampulla of Vater, stone in the ampulla or major ducts, and benign ulcer of the duodenum involving the ampulla.

Diseases in which pancreatic insufficiency most often occurs are cystic fibrosis of the pancreas and chronic relapsing pancreatitis. In chronic relapsing pancreatitis, the pancreatic insufficiency is usually a late manifestation.

SYMPTOMS OF PANCREATIC INSUFFICIENCY

The intent and purpose of this paper is primarily concerned with the insufficiency of the external secretion of the pancreas; therefore, pancreatic diabetes mellitus, a form of pancreatic insufficiency of

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From the Symposium on Diseases of the Pancreas, presented before the Sections on General Medicine, General Surgery and Radiology at the 81st Annual Session of the California Medical Association, Los Angeles, April 27-30, 1952.

the internal secretion, insulin, will not be included, except as it may be related secondarily to severe and extensive destructive pancreatic disease.

The symptoms of pancreatic insufficiency *per se* are those of indigestion and malnutrition owing to the lack of proper digestive enzymes and inadequate utilization of food materials. As was pointed out before, these inadequacies in slowly progressive disease may be so minor, or so long in reaching clinical proportions, as to remain unrecognized until the disease causing the insufficiency is well advanced. For these reasons the milder degrees of pancreatic insufficiency may be without symptoms, and then accompanied by vague and indefinite symptoms of indigestion long before the gross evidences of the nature of the insufficiency appear.

Acute pancreatic insufficiency accompanies complete, or nearly complete, obstruction of the pancreatic duct, such as may occur with impaction by stone, or compression by tumor or edematous inflammatory condition. The symptom of pain or painful discomfort owing to the obstruction so greatly overshadows the clinical evidences of the insufficiency of enzymes that the clinical features of insufficiency are unimportant except in the follow-up observations, treatment and prognosis.

The symptoms in chronic pancreatic insufficiency may be classified as general, digestive, nutritional, and metabolic.

The general symptoms appear only in the more profound and prolonged instances, and are manifest by weakness, fatigue, and gradual debilitation, probably related to the nutritional deficiency.

The digestive symptoms vary in degree and character, but include sense of abdominal fullness, especially in the upper abdomen, flatulence, anorexia, nausea and minor abdominal cramping or discomfort. Vomiting may occur. The tendency is toward constipation. Diarrhea or loose stools appear some time in the course of the symptoms, usually when steatorrhea and creatorrhea are well advanced. When the insufficiency is severe the stools are voluminous, fatty or greasy, rancid, and malodorous, characteristic of the indigestion of fat.

The nutritional and metabolic symptoms are related to the severity and duration of the insufficiency, and they vary from being unrecognizable to the profound symptoms of weight loss, liver functional impairment, hypoproteinemia, hypocalcemia, vitamin deficiency, and the vague painful discomforts which may be associated with osteoporosis. The inability to utilize fat and protein in particular accounts for the weight loss, weakness, and wasting. In the more severe instances there may be associated fatty change in the liver with impaired hepatic functions. Severe hypoproteinemia will be accompanied by the clinical features of this condition. Multiple vitamin deficiencies occur, and in particular the symptoms and physical findings of the deficiencies of vitamins A, B, D and K may be present, which will account for dryness of the skin, hyperkeratosis, glossitis, cheilosis, edema and neuritis, osteoporosis, and bleeding in the mucous membranes and skin.

If the disease of the pancreas has been severe and extensive enough to involve enough islet cells, diabetes mellitus may be a part of the picture of pancreatic insufficiency.

Pancreatic insufficiency, in itself, probably does not cause pain, and extensive and long standing pancreatic disease may not have associated pain. The most severe and characteristic pain of pancreatic disease is associated with obstruction of the pancreatic duct, or edema or hemorrhage into pancreatic tissue. However, the diseases of the pancreas which lead to pancreatic insufficiency are frequently enough the cause of pain that the discussion of symptoms would not be complete without some mention of the location and character of pancreatic pain. Indeed, the pain of pancreatic disease may lead to a diagnosis long before there is any clinical evidence of pancreatic insufficiency. In the diagnosis of chronic relapsing pancreatitis, one of the more common diseases with which pancreatic insufficiency may be associated, the most important symptom is the recurrent episodes of upper abdominal pain.[1, 2] This pain may be mild and last only a few hours, or it may be severe and prolonged. Usually it is located in the epigastrium or more generally in the upper abdomen. At times it may be more on the right side of the upper abdomen and extend through or around to the corresponding level in the back, which may simulate the pain of biliary colic. At other times it may be on the left side in the upper abdomen and extend to the corresponding level in the back, and this location is considered more characteristic. At still other times it may be described as band-like in the upper abdomen and back. It may shift in these different locations and, indeed, it may extend into or radiate into the anterior chest and shoulder regions. It tends to be gradual in onset and disappearance. The character of the pain may be steady or cramping, cutting, aching, boring, or burning. The recumbent position may aggravate the pain, so that the patient may prefer to sit up in a bent forward position.

The great variation in location and reference of pain probably is related to the extensiveness and location or distribution of the pathologic lesion in the pancreas. Bliss, Burch, Martin, and Zollinger[2] studied the localization of referred pancreatic pain induced by electric stimulation through electrodes placed in different parts of the human pancreas at the time of surgical operation for biliary disease.

They found that pain stimulus in the head of the pancreas localized in the epigastrium to the right of midline and was distributed from the sixth to the eleventh thoracic dermatome on the right; pain arising in the body of the pancreas localized in the mid-epigastrium and was distributed from the sixth to the eleventh thoracic dermatomes bilaterally; and pain from the tail of the pancreas localized in the left epigastrium and was distributed from the sixth thoracic to the first lumbar dermatomes on the left.

The high incidence of respiratory tract symptoms in children with chronic pancreatic insufficiency owing to fibrocystic disease of the pancreas should be mentioned. These symptoms are associated with bronchitis, bronchiectasis, or suppurative pulmonary disease. It has been thought that this vulnerability to respiratory infection is owing to nutritional deficiency; perhaps the keratomalacia of the bronchial mucosa associated with vitamin A deficiency is a factor in partial bronchial obstruction and concurrent infection. Andersen[1] reported upon 22 patients who, after early diagnosis, received dietary therapy. A special formula supplemented by vitamins and pancreatin was used and bronchial infection did not develop.

PHYSICAL EXAMINATION

Usually in milder and moderate degrees of pancreatic insufficiency no abnormalities are noted upon physical examination. When the insufficiency is severe and of long standing, the physical abnormalities are related to the secondary factors or complications of nutritional and vitamin deficiencies and metabolic disorders. Abdominal distention, with flatulence, is usual. The disease of the pancreas causing the insufficiency may be attended by abnormalities observable upon physical examination, such as general abdominal tenderness, especially in the upper abdomen, during and shortly after an acute episode of relapsing pancreatitis, or a palpable mass of tumor or cyst of the pancreas. It might be well to emphasize the importance of special effort to palpate the pancreas. Grott[5] described a method which may be helpful. With the patient in lateral position on his right side, the examiner stands in front of the patient and, with the left hand under the lower portion of the thorax, uses his right hand to press inward and downward until it reaches the left edge of the vertebral column. Then the procedure is repeated with the patient in the lateral position on his left side and the examiner using his left hand for palpatory maneuver. This permits the stomach and duodenum to be pushed forward and the examining hand can get more nearly over the space between the stomach and the vertebral column.

LABORATORY STUDIES

In general, laboratory tests of three types are employed in the study of pancreatic insufficiency; namely, (1) gross, microscopic and chemical examination of stools; (2) examination of duodenal juice obtained through duodenal intubation under fluoroscopic control; and (3) determination of response of lipase and amylase in the blood serum to provocative stimulation.

1. *The stool in pancreatic insufficiency*

Grossly the stool is bulky and light colored, and droplets of fat may be seen adherent to the fecal material, or may be seen floating if the feces is put in water. Microscopically, there is an increased amount of unhydrolysed fat, as demonstrated with Sudan 3 stain; and many undigested meat fibers with the transverse striations clearly visible in the muscle fibers. Chemically, there is pronounced increase in neutral fat and nitrogen. With the patient on a low fat diet, daily excretion of more than three grams of nitrogen suggests pancreatic insufficiency.

Determination of the fat-nitrogen content in the stool does not identify the causes of the steatorrhea or azotorrhea, but it is a measuring rod for the presence of either. Further studies will be necessary to evaluate the causes. Chemical studies of partition of fat may aid in distinction of idiopathic steatorrhea from pancreatic deficiency but the quantitation of pancreatic enzymes in the duodenum is more reliable.

2. *Examination of duodenal juice obtained by duodenal intubation under fluoroscopic control*

Even though this method is subject to all of the inaccuracies of any procedure of capturing the fluid contents in a hollow viscus by putting the end of a tube there under standardized circumstances, it provides the most reliable studies for directly defining or determining the degree of pancreatic insufficiency. The studies on the duodenal juice obtained include quantity, determination of bicarbonate content, and quantitation of pancreatic enzymes. Decrease in the enzymes is evidence of pancreatic insufficiency, and the disturbance in the normal range pattern of these several factors may aid in differential diagnosis as to cause. Frequently in chronic pancreatitis there is decreased concentration of enzymes, although early in the disease the volume may be normal. With progression of the disease, volume, bicarbonate content, and enzymes decrease.

3. *Response of lipase and amylase in the blood serum to provocative stimulation of the pancreas*

The agents used for stimulation are secretin, Urecholine,® neostigmine, or combinations of any of

these with morphine. The experience with these tests in the department of gastroenterology of Hahnemann Medical College points to the following deductions:

(a) In general, if the pancreas is normal there is no response to secretin, Urecholine, or neostigmine alone, but a prompt increase in serum content of lipase and amylase after combination with morphine.

(b) In general, in the presence of obstructed pancreas, without extensive acinous atrophy, there is an increase of 50 per cent or more (above a base line) in amylase or lipase content after the use of secretin, Urecholine, or neostigmine.

(c) In general, if there is extensive acinous atrophy of the pancreas no increase in serum content of amylase or lipase occurs after the use of secretin, Urecholine, or neostigmine, or any of them in combination with morphine.

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Telephone Technique

How is the telephone answered in your office? Brusquely and loftily, or pleasantly and with implication that the patient is important?

Thought given to telephone answering will be immediately helpful in your own relationship with patients, and ultimately in shaping the public attitude toward medicine. For example:

1. *Initial response to ringing telephone:*

WRONG: "Doctor's office."
"Just a minute, please."
"Hello."

RIGHT: "Doctor Jones's office, Miss Briarly speaking."
"Garfield 0-0000" (if there are several physicians using the same number).
"Doctors Jones and Black; may I help you?"

2. *On making appointments:*

WRONG: "I can give you Thursday at two."
"Doctor can see you Monday morning."
"Wednesday afternoon is the doctor's day off."

RIGHT: "Could you make it on Thursday at two?"
"Monday morning would be a good time if that is convenient."
"Doctor Jones will not be able to see you on Wednesday afternoon; could you come in Thursday?"

Pancreatitis and Carcinoma of the Pancreas

Some Aspects of the Pathologic Physiology

HUGH A. EDMONDSON, M.D., *Los Angeles*

• The physiological phenomena accompanying pancreatic disease in adults are related to the local and generalized reaction of the body to the blockage and/or leakage of the three enzymes—amylase, lipase and trypsin. The measurements of amylase and lipase in the serum are the most reliable criteria in the diagnosis of acute disease. Related changes may include hypocalcemia, hypopotassemia, hyperlipemia, hyperglycemia and decreased renal function.

In chronic pancreatitis, there is less fluctuation in the amounts of the enzymes in the blood. The presence of diabetes mellitus, demonstration of calculi by x-ray, and examination of the stools for excess fat and meat fibers are more important diagnostic guides.

In cancer of the pancreas, function tests using secretin stimulation of the gland followed by an examination of the external secretion or determination of the serum amylase have been used with some success.

THE MORE COMMON pancreatic diseases in adults, such as acute pancreatitis, chronic pancreatitis and cancer, are of most importance. Each of them may cause certain physiologic disturbances which can be measured by laboratory tests and are useful in the diagnosis and treatment of the disease and in determining the prognosis.

The pancreas produces about two liters per day of alkaline juice with a pH as high as 8.5,[17] rich in the enzymes amylase, lipase, trypsin and chymotrypsin. It is the effect of the blockage or leakage of these enzymes plus, in some instances, the destruction of the islets that produces a chain of physiological events which result in a pattern of characteristic departures from normal as determined by laboratory tests.

ACUTE PANCREATITIS

In acute pancreatitis there is the greatest variety of changes capable of measurement.[8] The escape of enzymes from the pancreatic ducts causes damage to tissue, edema, hemorrhage and fat necrosis. This results in local phenomena such as pain, tenderness, nausea and vomiting, segmental ileus and even visible subcutaneous hemorrhage in the flanks. Generalized changes may follow, such as hyperamylasemia, hyperlipasemia, hypocalcemia, hemoglobinemia, serum potassium abnormalities, shock and the alarm reaction.[8] Disturbance of renal function and lower nephron nephrosis have been noted.[8, 23] More recently, changes in the clotting mechanism of the blood have been recorded.[13]

The role of each of the enzymes as it infiltrates the tissue is important. Amylase appears to be innocuous. It is simply absorbed by the blood and eliminated in the urine. The most reliable basis for the diagnosis of pancreatitis is the presence of amylase in the blood and urine. Rarely is the test misleading, if other causes of hyperamylasemia are kept in mind. The other causes include perforation of the stomach or intestine, intestinal obstruction and infarction. In any of those conditions the amylase of the intestinal content can be absorbed but the content of the enzyme in the blood is nearly always less than 500 units, and usually under 300, per 100 cc.[8] Other abdominal conditions occasionally associated with an increase in the amount of amylase in the blood include acute cholecystitis,[18] appendicitis, retroperitoneal tumor[19] and volvulus of the colon. Extra-abdominal disorders such as myocardial infarction, pulmonary disease[28] and dissecting aneurysm may be accompanied by a rise in amylase values in the blood and occasionally have to be considered in differential diagnosis. In renal insufficiency the amount of amylase may rise because it cannot be excreted in normal amounts in the urine. This is especially true in uremia. The status of renal function should always be assayed even in the presence of pancreatitis as it may confuse the picture.

In recent years, another cause of high serum amylase and even of mild pancreatitis has been noted.[3] Morphine and codeine may apparently cause a spasm of the sphincter of Oddi of sufficient degree and persistence that dilatation of ducts and absorption of amylase occur. Even mild pancreatitis has been described in a patient with morphine poison-

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From the Symposium on Diseases of the Pancreas. Presented before the Sections on General Medicine, General Surgery and Radiology at the 81st Annual Session of the California Medical Association, Los Angeles, April 27-30, 1952.

ing. Further investigation may prove that some of the hitherto unexplained elevations in amylase content in the blood are owing to opiate therapy in such diseases as acute cholecystitis, dissecting aneurysm, myocardial infarction and pulmonary disease.

In mumps the salivary amylase is absorbed and the content in the blood may rise as high as it does in acute pancreatitis. As the nature of the disease is obvious, however, this causes no problem in diagnosis unless mumps pancreatitis is suspected. Then determination of the lipase and antithrombin content of the blood may be helpful.

It has been reported that in acute pancreatitis the rise of urinary diastase may lag as much as 24 hours behind the increase of amylase in the blood. Some data, including the author's, indicate this may be true; but in most patients the two are parallel in elevation and subsidence. If the diagnosis is in doubt, both the serum and urine should be tested repeatedly. Well controlled investigation by Dankner and Heifetz[6] indicated that if renal function is normal, the amylase is cleared promptly by the kidney. Dankner and Heifetz also observed that transient impairment of renal function is most likely to occur between 24 and 48 hours after the onset of the disease.

If the pancreas is completely destroyed the amylase values in the blood may be normal within the first 48 hours after onset. In such circumstances other tests may be used—determination of amylase content in peritoneal fluid removed by tap, and of the amount of calcium and antithrombin in the blood. Keith and co-workers[14] noted increased amounts of amylase in the peritoneal fluid for as long as three days after the serum value had returned to normal. They studied fifteen patients in this regard and in three cases no peritoneal fluid was obtained when a tap was made. In studying autopsy material, the author was surprised that the autopsy surgeon noted free fluid in the peritoneal cavity in only one-third of subjects with severe or fatal pancreatitis. The value of the peritoneal tap, when the diagnosis is in doubt, is further enhanced by examination of the fluid for bile pigment and acid content.

Lipase is responsible for fat necrosis, the characteristic gross finding in pancreatitis. It is absorbed and unusual amounts may be present in the blood for several days. According to Comfort and co-workers[4] it remains elevated even after the amylase content has returned to normal. The procedure for the determination of serum lipase is more difficult and time-consuming than that for amylase and therefore has not been so generally used.

The fatty acids formed when neutral fat is split by lipase combine with calcium to form soap. This in turn causes a drain on the serum calcium. In the author's experience, the serum calcium mirrors fairly well the quantity of fat necrosis. In severe damage it is below 8 mg. per 100 cc. of blood, and when below 7 mg. the outcome is usually fatal.[8] When depressed it may return to normal between the fourth and sixteenth day of the disease. Thus, determination of the calcium content of the blood may be helpful in the diagnosis in cases in which the patient is not observed until after the amylase content has returned to normal, or in cases in which there is complete destruction of the gland and amylase levels are normal or subnormal.

Perhaps the most interesting of all and the least understood is the action and fate of trypsin. The site of conversion of trypsinogen to trypsin is not known. But, when disseminated in the tissues, it is the apparent cause of hemorrhage[24] both by its direct necrotizing action on blood vessels and by its tendency to cause thrombosis of veins; thus adding to the anoxia of the tissues, furthering necrosis and obstructing the outflow of blood. Trypsin in proper concentration converts prothrombin to thrombin. This factor, added to the necrosis of vascular endothelium and consequent tendency to the deposition of platelets, would likely cause thrombosis. Partial to complete thrombosis of the pancreatic and splenic veins has been noted many times at necropsy.

Trypsin or trypsin plus lipase is also capable of causing hemolysis; thus hemoglobin may be released in the areas of hemorrhage and absorbed. Twice the author has noted a brown pigment in the serum of patients with severe pancreatic necrosis. One of them had severe oliguria, and lower nephron nephrosis was observed at necropsy.[8] Furthermore, in necropsy material the lesser omental cavity may be greatly distended with brown fluid while abundant gross evidence of hemosiderin is seen in the lining. There is reason, therefore, to believe that local hemolysis resulting in hemoglobinemia does occur in acute pancreatitis.

The fate of any trypsin absorbed by the blood is a problem all of its own. The plasma contains trypsin inhibitor,[25] a specific neutralizing substance which combines with trypsin. The author is now in the process of measuring both of these factors in patients with pancreatitis and various other diseases.

Innerfield and co-workers[13] produced hypertrypsinemia in dogs by the intravenous injection of the commercial trypsin. This caused a decrease in Ac-globulin, prothrombin and antithrombin for about five hours. Then Ac-globulin and prothrombin returned to normal, but the antithrombin rose to excessive levels for 15 to 17 hours. These investigators noted that in patients with acute pancreatitis the antithrombin remained high throughout the course of the acute illness. A similar rise in antithrombin did not occur in other acute abdominal conditions.

In a more recent report they stated that trypsin was present in the blood in each of the patients with pancreatitis.

The author has done the antithrombin tests* on a few patients. The content was normal in one with pancreatitis, increased in a patient with mesenteric thrombosis and also in one with metastatic carcinoma of the liver. It would seem that further experience with the antithrombin test is in order before it achieves the prominence given amylase in the diagnosis of acute pancreatitis.

Since 1846,[26] hyperlipemia with a milky serum has occasionally been noted in acute pancreatitis. This phenomenon is probably owing to more than one cause. Klatskin[15] observed that the course of idiopathic hyperlipemia may be complicated by attacks of acute pancreatitis, owing perhaps to clumped lipid particles in the vascular tree of the pancreas. Gardner[11] noted hyperlipemia in patients with high blood sugar values complicating acute pancreatitis. The lipemia disappeared with insulin therapy. The author has observed five instances of milky serum in pancreatitis. In three of the patients, no evidence of disturbed carbohydrate metabolism was noted nor was there a history compatible with idiopathic hyperlipemia. Careful laboratory study of the lipid problem was not carried out.

Fat embolism arising from the areas of fat necrosis may contribute to a fatal outcome in the disease.[8] In such circumstances fat stains of the lungs should be part of the histological study.

Hypopotassemia often occurs in the first 48 hours in severe pancreatitis.[8] Unless treated it does not return to normal until the patient is taking food by mouth. Several factors may contribute to the low serum potassium. The alarm reactions and loss by nasogastric suction are to be considered.

In severe necrotizing pancreatitis damage to the islets may produce diabetes mellitus,[27] even of such severity that acidosis supervenes. This possibility should not be overlooked as insulin therapy may prevent death.

CHRONIC PANCREATITIS

Recurrent attacks of acute pancreatitis often lead to chronic changes in the gland, but this is not invariably so. Occasionally a patient may have repeated attacks over many years and yet none of the characteristic symptoms of chronic relapsing pancreatitis intervene.

Early in chronic pancreatitis the amylase content of the blood may remain consistently elevated. Once fibrosis and atrophy have occurred in the pancreas, recurrent acute attacks may not be accompanied by such high serum amylase values as those ordinarily observed. Pseudocysts are common in chronic relapsing pancreatitis and may also cause persistently high amylase levels.

The more common physiological abnormalities in chronic pancreatitis are owing to the failure of the external secretion to enter the intestine in sufficient quantity to digest fats and proteins; steatorrhea and creatorrhea result. Contrary to the usual statement, failure of the internal secretion may occur fairly early in the course of the disease and diabetes mellitus will be the presenting symptom or the first complication noted after one or more attacks.[9] Glucose tolerance tests should be repeatedly done in patients with chronic relapsing pancreatitis.

The precipitation of calcium carbonate in the pancreatic juice to form calculi in the ducts and acini of the pancreas is another common complication. This, the author believes, is caused by the supersaturation of the juice with calcium and carbonate.[10] The demonstration of calculi by roentgenograms gives positive proof of chronic pancreatitis. In recent years, the author has noted calculi particularly in chronic alcoholics with pancreatitis which is often associated with cirrhosis.

Since the work of Agren and Lagerlof,[1, 2] many efforts have been made to measure the amount and concentration of enzymes in the external secretion as a guide to the progress of chronic pancreatitis.[19, 5, 21] Secretin, Mecholyl® or Urecholine® have been used to stimulate secretion. These methods require a tube with a double lumen, one for the duodenum and one for the stomach. This test is time-consuming and difficult for the patient, but valuable information can be gained from it.

Several investigators[16, 29, 12] have tried to arrive at the same conclusion by giving the patient a pancreatic stimulant and measuring the rise in serum amylase. There is a difference of opinion as to how helpful the results may be to the clinician. It seems safe to say that further investigation is necessary before a simple and highly diagnostic test for pancreatic function is evolved.

CARCINOMA OF THE PANCREAS

Physiological changes that aid in the diagnosis of cancer of the pancreas are few and vary with the location and extent of the tumor. Cancer of the tail of the pancreas usually does not affect the external or internal secretion. Cancer of the body of the gland may obstruct the duct of Wirsung or destroy extensively the secreting tissue. The same is true of cancer of the head of the pancreas, and in addition obstruction of the common duct occurs.

Fewer than half of the patients have increased content of amylase and lipase in the blood. If there is extensive destruction, diabetes may occur.

* Performed by Arnold G. Ware, Ph.D., and William H. Griffith, M.D., in the Department of Biochemistry at Los Angeles County Hospital.

Function tests using secretin, Mecholyl or Urecholine or some combination thereof have been attempted in the same manner as in the diagnosis of chronic pancreatitis. The most valuable test seems to be determination of the response of the external secretion to secretin given intravenously. Dreiling,[7] using this test, noted there was a decreased volume of juice in a high percentage of patients with cancer of the head and/or body of the pancreas. Cancer of the body with great destruction caused a pronounced decrease in the concentration of amylase and bicarbonate as well as small volume.

The serum secretin test,[16, 29] in which the amount of amylase in the blood is determined before and after injection of secretin, resulted most often in a failure of the amylase to rise. However, in some patients, presumably those with obstruction near the head of the pancreas, the serum amylase increased sharply.

Duodenal drainage has an advantage over the serum secretin test in that the bile pigments can be studied at the same time. Because secretin is an efficient cholagogue, the gallbladder empties. Thus, in jaundiced patients a normal flow of pancreatic juice in the absence of any biliary pigment would place the obstructive lesion in the biliary tract.

The early diagnosis of cancer of the pancreas remains a difficult problem that awaits more discerning methods.

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Adenoma of the Islets of Langerhans

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• *An adenoma of the islets of Langerhans may be found anywhere in the pancreas but is more common in the body and tail, and more than one tumor may be present. When such a tumor produces insulin the symptoms observed are those of hyperinsulinism, varying from pallor, sweating, weakness and amnesia in mild cases to coma and convulsions in the more severe.*

The diagnosis rests largely on Whipple's triad, namely, the onset of symptoms during the fasting period or after exertion, sugar content in the blood of less than 50 mg. per 100 cc. during an attack, and immediate relief of symptoms upon the administration of glucose in one form or another.

Surgical removal of an adenoma is the only known cure, and unless it is done the patient will eventually die of the effects of prolonged hyperinsulinism. The procedure entailed may be relatively simple, or fraught with great difficulties and postoperative complications of a major nature.

The literature indicates that there has been a mortality rate of 8 or 9 per cent associated with the surgical treatment of this condition.

WHEN Banting and Best[1] in 1922 discovered insulin, the long sought hormone produced by the islets of Langerhans, the discovery was not only a boon in the treatment of diabetes mellitus, for it was quickly to become the basis for one of the most dramatic and interesting chapters in the history of surgery. One year after Banting and Best's discovery, Harris[5] suggested the possibility of the occurrence of hyperinsulinism, analogous to hyperthyroidism, as the cause of hypoglycemic symptoms in persons not taking insulin. This hypothesis was proved correct in 1927 when Wilder and co-workers[12] reported the first case of hyperinsulinism associated with a tumor of the pancreas. The tumor was a carcinoma of the islets of Langerhans with metastasis to the liver, and from one of the metastatic nodules was obtained a substance which proved to have an insulin-like action when injected into rabbits. In 1929 Graham[7] removed from the pancreas of a patient with symptoms of hypoglycemia a small, circumscribed tumor which was considered to be a slowly growing carcinoma of the islet tissue. The operation was followed by complete relief from the symptoms; it effected the first known cure of hyperinsulinism caused by a tumor of the islets. Howard, Moss and Rhoads[6] in a recent review of reports of 398 cases noted that in 313 cases (78.6 per cent) the lesion was described as benign adenoma, in 37 (9.3 per cent) as carcinoma and in 48 (12.1 per cent) as tumors in which malignant change was suspected. However, only 200 of 361 patients with localized tumors were operated upon for hyperinsulinism. In the other 161 the tumors were not observed until autopsy; in some instances the tumors had been "missed" at operation, in others they were observed in the bodies of patients who had died from undiagnosed hyperinsulinism, and in some cases were noted incidentally in other autopsy observations. A tumor was observed at the time of operation in only 82 of the 200 cases in which exploration was carried out. Of the 118 patients in whom no tumor was found at exploration, 37 eventually were found to have an islet-cell tumor; in 12 of the 37 cases the adenoma was resected blindly during subtotal pancreatectomy, in 12 the tumor was found and removed at a subsequent operation, and in 13 the tumor was not found until autopsy. On the other hand in 81 patients an islet-cell tumor was never observed. Fifty-six of the patients in that group had partial pancreatectomy; four of them died of the operation and 26 had satisfactory therapeutic result. The data indicate that in many cases both diagnosis and treatment are beset with difficulties and that the degree of success achieved thus far in these aspects of islet-cell adenoma leaves room for considerable improvement.

Reporting upon autopsy studies, Lopez-Kruger and Dockerty[8] noted that only in about one case in five does an islet-cell adenoma produce insulin in amounts large enough to cause symptoms of hypoglycemia. It is known that different types of cells, the counterparts of those found in normal islet tissue, are present in an adenoma, and while many authorities are of the opinion that insulin is produced by the cells of the beta type, yet in histological studies of insulin-producing (functioning) and non-insulin-producing (non-functioning) adenomata the answer to why some adenomata produce insulin while others

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do not has not yet been found. Any symptoms to which a non-insulin-producing adenoma may give rise will only be those produced by the tumor *per se;* and as most such adenomata do not reach sufficient size to cause any symptoms at all, they are usually found incidentally, most frequently at autopsy.

Assuming that the amount of islet tissue in a normal pancreas represents about 2.5 per cent of the whole gland, the presence of a functioning adenoma weighing 1.5 grams would increase the insulin-producing tissue by nearly 70 per cent. No wonder, then, at the havoc such tumors cause in the sugar content of the blood.

Hyperinsulinism owing to an islet-cell adenoma occurs most commonly in the fifth and sixth decades of life, rarely later. The incidence is slightly higher in males than in females.

Approximately 70 per cent of islet-cell adenomata are situated in the body or tail of the pancreas. In about 12 per cent of cases more than one tumor is present—usually not more than two, but as many as five have been found.

Adenomata at this site vary greatly in size. Some are only 1 mm. in diameter. The largest reported was 15 x 13 x 10 cm. and weighed 673 gm.[2] It was an insulin-producing tumor but, despite its size, caused hyperinsulinism of no greater degree than that produced by much smaller adenomata. The author knows of no case of hypoglycemic symptoms owing to adenoma of the islet-cells in which the tumor was less than 1 cm. in diameter.

SYMPTOMS AND DIAGNOSIS

The time-tested triad postulated by Whipple[11] as diagnostic of the presence of an islet-cell adenoma needs little or no modification. The onset of symptoms is always during the fasting period or after exertion. The symptoms are referable chiefly to the nervous system; they may vary in intensity from minor mental aberrations and amnesia resembling petit mal to unconsciousness and convulsions more violent than those of grand mal. Usually the content of sugar in the blood during an attack is less than 50 mg. per 100 cc. However, the fact that sugar content quite as low has been observed in patients with islet-cell adenoma at times when there were no such symptoms present, indicates that the rapidity of decrease in the sugar content may play some part in determining the time of onset of an attack. While ordinarily the administration of glucose effects immediate relief of symptoms, it should be borne in mind that in rare instances, such as that recorded by Mitchell and co-workers,[9] it may fail to do so, probably because of irreversible changes in the central nervous system produced by the oft-repeated hypoglycemic state.

There seems to be no unanimity of opinion regarding the merits of the intravenous glucose and intravenous insulin tolerance tests to determine the presence of hyperinsulinism, but in two cases observed by the author the results of the latter test were in accord with Collip's[4] postulate and thus proved of definite value.

In considering differential diagnosis, it would appear that other causes of hypoglycemia (except hyperplasia of the islet tissue itself), such as diseases of the pituitary gland, adrenal glands, thyroid gland or liver, are unlikely to produce the profound degree of hypoglycemia characteristic of islet-cell adenoma; and in the event disease of these other glands were sufficiently advanced to give rise to such severe hypoglycemia, it is probable that the etiological factor would be apparent.

Roentgen studies after a barium meal may be of help in diagnosis if the adenoma is big enough or is in such a position as to cause deformity of the duodenal loop. Using the more exact Engel-Lysholm technique for roentgen examination of the pancreatic bed, Olsson[10] observed an adenoma only 1.5 cm. in diameter situated on the anterior surface of the pancreas.

TREATMENT

The only hope of cure lies in removal of the adenoma. As the tumor may be in any portion of the gland, the incision must be such as to permit adequate exposure of the entire pancreas. It may be either vertical or transverse, although most surgeons appear to favor the latter. The gland is approached through the gastrocolic omentum between the stomach and the transverse colon. Sometimes the adenoma or a localized swelling produced by it may be seen as soon as the pancreas is adequately exposed, or a localized tumor, firmer than the gland tissue, may be readily felt on palpation. The finding of a tumor, however, must not deter the surgeon from painstaking search of the remaining portion of the gland, as more than one adenoma may be present. Moreover, as tumors may also occur in aberrant pancreatic tissue, the immediate environs of the pancreas, at least, must be examined carefully. In the event no tumor can be found, there are two possibilities to consider: partial pancreatectomy in which the body and tail of the gland are removed, and total pancreatectomy. It is to be emphasized, however, that neither partial nor total pancreatectomy should even be considered until after the head of the gland has been thoroughly examined; and examination of the head of the pancreas cannot be considered thorough unless the duodenum is mobil-

ized and the gland tissue gently freed enough to permit of palpation of it between the thumb and finger. Not all tumors in the head of the pancreas can be felt by palpation through the anterior surface alone, as postmortem evidence attests.

If resection is deemed necessary, choosing between partial and total pancreatectomy involves the following considerations:

Partial pancreatectomy. (1) It is more likely that the tumor is in the body or tail of the gland. (2) Removal of those portions does not entail the interference with the biliary and gastrointestinal tracts that is necessary in removal of the head. (3) There is always a possibility, slight though it may be, that the lesion present is hyperplasia of the islet tissue, rather than an adenoma. If evidence of hyperplasia is noted when pathologic examination of the removed body and tail is carried out, the surgeon has grounds for hope that the operation done was extensive enough at least to relieve the symptoms if not effect complete cure.

Total pancreatectomy. The operation is one of greater magnitude and it entails trauma to the biliary and the gastrointestinal tracts. However, when an adenoma has not been found in careful examination of the pancreas, the only way to make certain that a tumor is not left behind is to remove the head of the gland as well as the body and tail. Physiologic disturbance following total pancreatectomy is relatively slight.

Pancreatic operations, sometimes relatively simple, may be exceedingly difficult, and the likelihood of complications following operations upon this gland appears to be definitely greater than that experienced in connection with operations upon other abdominal viscera. Even enucleation of an islet-cell adenoma is not without risk; a mortality rate of 8 or 9 per cent has been associated with that procedure.

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Standard of Living

"People who are to be comfortable are accustomed to lie on sofas, and dine off tables, and they should have sauces and sweets in the modern style." . . .

"Then we must enlarge our borders; for the original healthy state is no longer sufficient. Now will the city have to fill and swell with the multitude of callings which are not required by any natural want. . . . And living in this way we shall have much greater need of physicians than before?"

"Much greater."

—*Plato's Republic*, translated by Benjamin Jowett (World Publishing Co., 1946)

Operations on the Pancreas

Some Technical Considerations

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• Many diseases of the pancreas formerly universally considered fatal, now are known to be amenable to surgical therapy. Pancreatic cysts, pseudocysts, calculi, inflammation, and benign and malignant tumors all can be dealt with effectively by operation. In this presentation various surgical techniques which have proved to be of therapeutic importance are considered.

CONSIDERED from a surgical point of view, the pancreas is intimately related anatomically with neighboring organs and contiguous structures, such as the duodenum, the spleen, the common bile duct and the superior mesenteric and splenic vessels. At laparotomy it is obscured from view by the stomach, the gastrocolic omentum and the colon. The uncinate process almost completely surrounds the superior mesenteric vessels, while the lower end of the common duct usually traverses the head of the pancreas before entering the duodenum. Because of these close anatomical relationships, surgical operations upon the pancreas are not simple. They are usually time-consuming and may become extremely hazardous, especially in relation to the possible danger of injury to neighboring structures. Serious hemorrhage as well as persistent duodenal, bile or pancreatic fistulae are the well known consequences of faulty operative techniques.

Probably the most common lesions of the pancreas of surgical importance are acute and chronic inflammations. Many different surgical procedures, none universally successful, are currently employed in the treatment of pancreatitis. Most of them are based upon the concept that the disease is caused by increased pressure within the pancreatic ducts, presumably owing to obstruction of the main pancreatic duct, with continued secretion of pancreatic juice, or to obstruction in the common bile duct with reflux of bile into the pancreatic ducts.

Now that it has become recognized generally that laparotomy with incision and drainage of the pancreas is not effective treatment for acute pancreatitis of either the edematous or the acute hemorrhagic type, the acute stages of the disease, when recognized, are treated conservatively. Except in mild edematous pancreatitis, shock and rapid circulatory failure are early manifestations and must be treated vigorously. Surgical intervention at this stage causes death in a relatively high proportion of cases, and as the symptoms are much like those of other acute upper abdominal lesions which require early operation, correct differentiation becomes imperative. An elevation in serum amylase is an invaluable diagnostic aid.[7]

Although some surgeons have advocated T-tube drainage of the common duct in the acute stage of pancreatitis, evaluation of its effectiveness is difficult. Most surgeons agree that medical management in this stage is more effective. Splanchnic block with procaine is said to relieve the pain of an acute attack and to cause relaxation of the sphincter of Oddi, thus diminishing back pressure in the pancreatic ducts.[9] Here again it is difficult to know how long a given attack might have persisted without this added treatment. The author believes that most acute attacks of pancreatitis of the edematous type will subside spontaneously and that the acute hemorrhagic form will cause death in a high proportion of instances regardless of the kind of treatment.

Relapsing pancreatitis or recurring subacute pancreatitis has been treated by a number of procedures directed toward either improving drainage from the pancreatic duct or reducing pancreatic secretion through the reduction of gastric secretions. DeTakats and Walter[3] expressed belief that vagotomy or subtotal gastric resection may sufficiently decrease gastric secretions. Results from the various procedures have not been encouraging.

Chronic biliary tract disease is frequently associated with relapsing pancreatitis. Often cholecystectomy or choledochostomy for correction of the former will also relieve the latter. Prolonged drainage of the common bile duct has proven to be a very useful procedure. This can be accomplished either by T-tube drainage or by cholecystojejunostomy or choledochojejunostomy. Ascending infection of the biliary tract is liable to occur, however, unless anastomosis is made into a defunctionalized loop of

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bowel. Anastomosis to an intestinal limb of Roux-Y type or to a long loop of small bowel shunted with an entero-entero anastomosis tends to prevent ascending infection.

Many physicians have directed treatment toward relief of spasm or other kinds of obstruction of the sphincter of Oddi.[3] Archibald[2] as early as 1913 suggested division of the sphincter of Oddi. Doubilet and Mulholland[4] devised a special instrument which may be passed through the common duct into the duodenum, severing the sphincter of Oddi. Some surgeons have reported good results from transduodenal sphincterotomy.

Interruption of the autonomic sensory fibers by various procedures has been proposed not only as a means of controlling spasm of the sphincter but also to relieve pain, just as in the acute state of the disease. Some investigators have reported good results with simple unilateral splanchnicectomy while others have proposed more extensive procedures such as bilateral dorsal sympathectomy, splanchnicectomy and vagotomy.

Chronic pancreatitis, pancreatic calcinosis and pancreatic lithiasis are so frequently associated one with the other that they may be considered together in discussion of treatment. Intelligent surgical management of any of the conditions depends upon the clinical manifestations and the localization of the process. Rarely, a stone obstructing the main pancreatic duct can be removed by direct incision into the duct, followed by T-tube drainage.[5] Usually calcification is diffuse throughout the gland and nothing short of removal of all or part of the pancreas will suffice. When the disease process is confined to the head or tail of the pancreas, it may be successfully treated by partial excision of the gland. Usually, the disease involves so much of the gland and destruction is so extensive that there is little if any gland to be preserved. In such circumstances treatment is directed toward the control of pain. Total pancreatectomy followed by substitution therapy has been successful in a few cases. However, pancreatectomy for the relief of extensive pancreatitis with or without calcinosis is an extremely hazardous procedure, owing mainly to the danger of uncontrollable hemorrhage in the extremely vascular field.[13]

Neoplasms of the common duct, of the ampulla of Vater, of the head of the pancreas and of the duodenum involving the pancreas all lead to jaundice. The interest of many surgeons in recent years in problems associated with the surgical treatment of these conditions has led, in a large measure, to present concepts of operations on the pancreas. Means of controlling hemorrhage in jaundiced patients, control of infection, safe anesthesia for prolonged periods and meticulous surgical technique have made extensive resections of this region relatively safe and in certain conditions very much worthwhile. Often the major problem at the time of operation is the sometimes exceedingly difficult one of differentiation between stone in the common duct, chronic pancreatitis and periampullary carcinoma. Biopsy with a frozen section is not only apt to be misleading but can lead to serious postoperative complications. Usually clinical impression must be relied upon in determining the course of therapy.

The operability of malignancies in this region is often difficult to determine. Invasion of the tumor into the superior mesenteric vessels makes radical pancreaticoduodenectomy impractical. Although it is stated that the best way to determine whether or not such invasion is present is to reflect the duodenum and head of the pancreas medially, in the author's experience the uncinate process is usually so large that it surrounds the superior mesenteric vessels, both laterally and posteriorly, and reflection of the duodenum and pancreas only displaces these vessels and does not expose them. By transecting the gastrocolic and gastrohepatic omentum the junction of the body and head of the pancreas can be exposed and by gentle blunt dissection of the pancreas from its inferior and superior margins along the course of the mesenteric vessels, it is possible to determine whether there is fixation of a tumor mass to these vessels. In the author's opinion, other contraindications to resection are distant metastasis, irresectable local extension of tumor, and involvement of the portal vein with tumor.

If radical pancreaticoduodenectomy is undertaken, the most simple and most rapid reconstruction is important. The anastomosis of the bile duct to the small bowel should be at a point proximal to the gastroenterostomy, as should the anastomosis of the pancreatic stump. This is accomplished best by resecting the entire duodenum to a point beyond the ligament of Treitz. The jejunum then may be brought up anterior to the transverse colon and anastomosed end-to-end to the open end of the dilated common duct. If too great a discrepancy in size exists, an end-to-side anastomosis may be made and the open end of the jejunum closed. The stump of the pancreas is anastomosed end-to-side to the jejunum after the open end of the pancreas is closed with interrupted silk sutures. The pancreatic duct is permitted to protrude beyond this closed end. An incision is made in the wall of the jejunum through the serosa and muscularis just long enough that the closed end of the pancreas will fit into it. As these two structures are approximated, the protruding pancreatic duct is introduced into the lumen of the jejunum through a puncture wound in the mucous membrane. The duct and mucous membrane are tacked together with one or two fine catgut sutures. The serosa of the bowel is sutured then to the cap-

sule of the pancreas with interrupted silk sutures, thereby jutting the closed end of the pancreas against the outer surface of the mucous membrane of the bowel. The open end of the stomach then is sutured end-to-side to the jejunum. With this method there are only three suture lines, and as the bile duct and pancreas empty into a defunctionalized segment of bowel well above the gastroenterostomy, reflux of gastrointestinal contents into the biliary or pancreatic tree is prevented.

The utilization of this standard procedure for all conditions requiring resection of the head of the pancreas and duodenum has led to a surprisingly low operative mortality rate and a minimum of postoperative complications.

The successful management of cysts of the pancreas depends upon the underlying pathological condition which has led to the development of the cyst. True cysts are lined with epithelium and may be divided into three main classes: Retention cysts, congenital cysts and proliferative cysts. Pseudocysts are localized collections of fluid secondary to traumatic rupture or inflammation within the gland. The surgical procedure to be employed is dependent upon the type of cyst encountered, at operation. Because proliferative cysts are true neoplasms comprising the cystadenomas and cystadenocarcinomas, they must be extirpated.[12] Incision through pancreatic tissue beyond the gross limits of the tumor may be necessary to find a proper cleavage plane and to insure a margin of normal pancreas about the excised tumor mass. At times it is necessary to remove entire portions of the pancreas to insure complete extirpation of a potentially malignant cyst.

In contrast to proliferative cysts, pseudocysts of the pancreas occur quite frequently and may be treated by marsupialization and drainage, extirpation or internal drainage. Excision is rarely possible because of the fixation secondary to the inflammatory reaction which accompanies their formation. Dissection is difficult because it jeopardizes such important structures as the common duct and superior mesenteric, hepatic and splenic vessels, as well as the portal vein.

The major objection to marsupialization[10] is the persistence of a draining sinus tract. Digestion of the abdominal wall about the cutaneous opening may be expected and the condition is difficult to manage. Other complications are secondary infection, hemorrhage and, often, pronounced debility.

Internal drainage as a method of treatment not only eliminates the fistula formation of external drainage but is effective and is not as formidable a surgical procedure as complete excision. Various channels of drainage have been used—anastomosis of the cyst to the stomach, to the duodenum, to the jejunum and to the gallbladder—with varying degrees of success. Experience seems to have proven that internal drainage is best accomplished by anastomosis between the cyst and a defunctionalized loop of jejunum. This is accomplished either by the employment of a long loop of jejunum isolated by an entero-entero anastomosis or best by drainage into the defunctionalized limb of a Roux-Y intestinal anastomosis. The prevention of regurgitation of gastrointestinal contents into the cyst is important. The inflammatory, fibrous lining of a pseudocyst facilitates its obliteration after prolonged drainage.

Islet cell tumors of the pancreas may produce hyperinsulinism, causing the syndrome well known in association with insulin shock. Since W. J. Mayo's report of a malignant islet cell tumor in 1927 and Graham's report of a permanent cure of symptoms of hyperinsulinism following removal of a benign adenoma of islet cell tissue in 1929, a number of such cases have been recorded.[6] It is now recognized that surgical operation is the treatment of choice. Finding the tumor at the time of operation is often extremely difficult. It may be very small and of the same color and consistency as the remaining pancreatic tissue. Sometimes more than one tumor is present. For these reasons the entire pancreas must be exposed and adequately explored. The tumor when found is excised. When no tumor is found, resection of the body and tail (where about 80 per cent of the growths arise) and immediate inspection by a competent pathologist is advisable. If a tumor is not found in the excised portion, the head of the pancreas must be resected.

The results when the tumor is located and removed are excellent. Occasionally, good results follow extensive resection, even though no tumor is observed in the specimen. Only a small percentage of pancreatic tumors are malignant.

Annular pancreas, an exceedingly rare condition, may occasionally lead to chronic duodenal obstruction owing to the growth of a ring of pancreatic tissue around the second portion of the duodenum. The symptoms are best relieved by by-passing the process rather than by operation upon the gland because of the danger of injury to the pancreatic ducts. Gastric resection is indicated when the condition is complicated by gastric or duodenal ulcer.

Injuries to the pancreas caused by either concussive or penetrating wounds of the abdomen usually require immediate surgical management.[8] Pancreatitis secondary to blunt trauma often is unrecognized as such at the time of injury and may therefore lead to subsequent development of pancreatic pseudocyst. Penetrating wounds of the abdomen rarely involve the pancreas alone. The injury to the pancreas is usually discovered in association with penetrating injury to surrounding viscera. Complicated injuries

of this kind must be dealt with as indicated by conditions present at the time of operation. Careful repair of lacerations with ligation of the injured ducts should be carried out when possible. Resection of badly damaged pancreatic tissue often will prevent serious postoperative drainage and digestion. Proper drainage must be instituted if leakage of pancreatic secretions appears likely.

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Use of Gamma Globulin in Poliomyelitis

WHETHER GAMMA GLOBULIN will be effective in the prevention of paralytic poliomyelitis is not now known. On the basis of animal experiments and preliminary study on humans, it is possible that globulin will have value in human poliomyelitis, but serious questions remain to be answered before such a hope can be substantiated. Nevertheless, public dissemination of information on the status and objectives of current studies, incompletely presented or misunderstood, has created a serious demand for gamma globulin which cannot be met.

Virtually the entire output at current production rates is required to meet the demand for prevention or modification of the course of measles and infectious hepatitis.

Under the circumstances, it is obvious that the existing limited supply and current production of gamma globulin should be reserved for use in these diseases in which its efficiency has been established.

—Statement supplied by the chairman of the subcommittee on blood of the Health Resources Advisory Committee.

Coxsackie Virus in Southern California

Isolation of a Strain from Stools of a Patient

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• *Thirty-three stool specimens from 29 patients were examined for Coxsackie virus by the inoculation of suckling mice. Such a virus, designated "California I," was obtained from two stool specimens collected on successive days from a patient with so-called nonparalytic poliomyelitis.*

Neutralizing antibodies for the California I strain of Coxsackie virus could not be demonstrated in serum obtained from the patient early in the illness, but were present in convalescent serum.

Serum from the patient's daughter, who previously had had a similar illness, neutralized the strain of virus isolated from the father.

In pathologic examination of the skeletal muscles of mice infected with the California I virus, lesions typical of those produced by Coxsackie virus, group A, were noted.

California I strain of the virus was not neutralized by immune serum prepared from several other strains of Coxsackie virus.

WHILE STUDYING several small epidemics of poliomyelitis in upstate New York in 1947, Dalldorf and Sickles[3] isolated a virus by the intracerebral injection of suckling mice and hamsters with supernates from fecal specimens obtained from two children living in Coxsackie, N. Y. The virus, now designated "Coxsackie virus," produced paralysis and death in the mice and hamsters. Upon pathologic examination widespread degeneration of the skeletal muscles was observed. In the same year, in Wilmington, Del., an outbreak of atypical poliomyelitis occurred in which multiple infections were observed in families and the paralysis was transient in many cases. Furthermore, several investigators failed to isolate the classic virus of poliomyelitis. Dalldorf[4] examined 21 specimens of feces from patients in the area and in five instances again recovered a virus that produced paralysis and death in suckling mice.

During the years 1947, 1948, and 1949, Dalldorf and Gifford[5] examined 517 stool specimens from patients in large and small epidemics of poliomyelitis and from a few sporadic cases. Coxsackie virus was obtained from 30 (5.8 per cent) of the specimens. Dalldorf[6] observed that most of the strains of virus produced extensive generalized degeneration of striated muscles in mice, and a few strains caused, in addition to the muscle degeneration, lesions in the central nervous system. Those that caused only muscle degeneration were classified as group A viruses, and those that caused lesions in the central nervous system were classified as group B. In 22 of the previously mentioned 30 cases the viruses isolated were observed to be group A, and some paralysis had developed in 14 of the patients. In the remaining eight cases the viruses were group B, and none of the patients was paralyzed.

Melnick, Shaw and Curnen, [13, 16] isolated a similar virus from patients. They detected Coxsackie viruses in sewage and flies and described cases of the disease among laboratory personnel working with the virus. Subsequently, the virus was isolated in other areas of the United States,[2, 8, 9, 11, 15] also in Canada,[1, 9] Denmark[12] and England.[7] Slater and Syverton[17] in 1950 isolated Coxsackie virus from a patient with nonparalytic poliomyelitis in Minnesota and maintained the virus in tissue culture for 24 successive passages.

Coxsackie virus produces a variety of symptoms; namely, fever, headache, backache, muscle pain and spasm, stiff neck, abdominal pain, nausea, vomiting, pleural pain, sore throat, malaise and anorexia. The most consistent symptoms are fever and headache, yet they vary in different epidemics. Paralysis occurs in a small percentage of cases. The clinical laboratory findings are limited chiefly to pleocytosis of the cerebrospinal fluid. Two rather distinct clinical syndromes, other than that resembling poliomyelitis, have been associated with the virus. Findlay and Howard[7] in England recovered the virus from patients with so-called epidemic pleurodynia, which has been known as Bornholm disease. Hueb-

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ner and co-workers[10] reported isolation of group A Coxsackie virus from 32 patients with herpangina in Maryland.

Coxsackie virus has been detected in several areas of the United States, but to date it has not been reported in the southwestern states with the exception of Texas. The present study was undertaken to determine if the virus could be detected in California. Poliomyelitis is endemic in the Los Angeles area throughout the year and the so-called nonparalytic form of the disease is prevalent.

MATERIALS AND METHODS

Stool specimens and paired blood samples were collected during the months of May, June, July and August 1951 from 29 patients at the Long Beach Veterans Administration Hospital with tentative diagnoses including septic meningitis, nonparalytic poliomyelitis, pneumonitis, and ulcerative colitis. The sera for neutralization tests and the stool specimens were stored at —70° C. The inoculum was prepared from the stool specimen, after thawing, by making a 20 per cent suspension in distilled water. The suspension was ground in a Ten Broeck grinder and centrifuged in a Spinco for one-half hour at 13,000 rpm. The supernate was exposed for one-half hour at room temperature to 1,000 units of penicillin and 10.0 mg. of streptomycin per milliliter. Three-hundredths of a milliliter of the inoculum was injected either intramuscularly or intraperitoneally into mice from one to two days old. The mice were observed at frequent intervals and whenever paralysis appeared they were removed and immediately frozen at —70° C. Inoculated mice that had nervous symptoms or that appeared unthrifty were sacrificed and washed with ether. Subsequently, the feet, tail, head, skin and viscera were removed and the remaining skeletal tissue finely minced for repeated animal passage. A 20 per cent suspension of tissue in distilled water was prepared in a Ten Broeck grinder, and centrifuged at 1,000 rpm. in a cold room for five minutes. Groups of suckling mice were injected either intramuscularly or intraperitoneally with 0.03 ml. of the supernate. In a few instances, blind passages were repeated several times.

Identification of the virus was attempted by neutralization tests using immune sera prepared from known strains of Coxsackie virus. The immune serum was produced by inoculating three-week-old mice intramuscularly or intraperitoneally with 0.1 ml. of a 10 per cent suspension of infected muscle-bone. Two inoculations were made each week for three weeks. After a rest period of one week, a single inoculation was made. The mice were bled two weeks later. The neutralization tests described by Melnick and Ledinko[14] were carried out.

The suckling mice were obtained from two separate colonies of white mice. They were pooled from several litters and redistributed to the mothers to avoid the use of one litter for a single inoculum. The inoculated mice were isolated in special quarters distant from the normal mouse colony and from the laboratory in which mice infected with known strains of virus were housed. At various times, litters from the two normal mouse colonies were tested for Coxsackie virus and always found to be negative.

RESULTS

The inoculum from only two of 33 stool specimens produced paralysis in suckling mice. The two specimens were obtained from the same patient on the sixth and seventh days of illness. All of a group of ten mice injected with inoculum from the first specimen, and all of a group of six given inoculum from the second specimen had paralysis in five days. Inoculum from a third specimen obtained from the patient 20 days after the onset of illness did not produce paralysis in a litter of six one-day-old mice.

The patient from whom the specimens were obtained was a 32-year-old white male cement mill operator. He was well until the day before admission to the hospital. The illness began with fever, chills, headache and backache, with stiff neck, nausea and vomiting on the second day. The acute illness continued for five days with fever as high as 103.6° F. Pain in the chest was present during the first week of illness. Nuchal rigidity persisted for 14 days. During the first two weeks of illness there were from 75 to 750 lymphocytes per cu. mm. of cerebrospinal fluid. The patient was discharged 19 days after admission to the hospital and remained well. As the patient had said that his eight-year-old daughter had likewise been ill with similar symptoms—sore throat, fever, headache, stiff neck—three weeks before he himself had become ill, serum was obtained from her and it neutralized the virus isolated from the father.

The virus isolated from the two stool specimens from the patient on successive days was not neutralized by immune sera prepared from Dalldorf, type I, II, III, Highpoint, Texas, and Connecticut 5 strains, but was neutralized by a homologous antiserum. Serum obtained from the patient early in the illness was ineffective but serum collected 20 days after the onset of the disease neutralized the virus. The virus has been designated as "California I."

In a pathologic examination of the skeletal muscles of mice infected with the California I strain, extensive degeneration was observed. The lesions were typical of those produced by group A Coxsackie virus. No lesions were noted in the tissues of the central nervous system.

DISCUSSION

Although a virus, identified as of Coxsackie type, was isolated from only two of 33 stool specimens obtained from 29 patients, it should be pointed out that the patients were adults ranging in age from 28 to 60 years, an age group older than that in which the virus is most prevalent. In the series reported upon by Dalldorf and Gifford,[5] 73 per cent of the patients were under ten years of age. Although no reports have appeared in the literature that Coxsackie viruses have been isolated in California, they would be expected to be as prevalent here as elsewhere. Virologists have not made a diligent search. The constant endemicity of poliomyelitis in Southern California is well known. Inasmuch as one form of illness resulting from infection with Coxsackie virus is similar to poliomyelitis, it may, in some cases, be erroneously diagnosed as poliomyelitis. The differentiation of the two diseases is dependent upon the intracerebral inoculation of monkeys to determine whether the virus of poliomyelitis is or is not present.

In the case of the patient from whom California I virus was obtained, it is probable that the patient's daughter had been infected by the same virus three weeks earlier than he, inasmuch as she had similar symptoms and serum obtained from her neutralized the virus obtained from him.

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Abscesses of the Breast

Recurring Lesions in the Areolar Area

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• Subareolar abscesses beginning either in infected skin glands or in breast ducts have an extraordinary tendency to recur and to be resistant to treatment. About three-fourths of 64 patients observed had from one to many recurrences of abscess after either spontaneous or surgical drainage, and many even after wide excision of scar in an interval of quiescence.

The most successful of a number of methods of treatment used was wide removal of scar and underlying chronic abscess cavity combined with removal of the ampulla and mouth of a connecting duct.

In a substantial number, after either drainage or unsuccessful excision, the process gradually subsided over a period of months or years.

Cancer has not been observed in any of the 64 patients.

ACUTE INFECTIOUS MASTITIS in the non-lactating breast is much more common than is generally recognized. Probably in most cases the infection arises in the stagnant fluid of a cyst or a dilated duct. Many of these lesions subside spontaneously with complete disappearance of any lump that had been palpable at the height of the inflammation. When such a lesion does point and drainage is necessary, if it is located away from the areolar area it commonly behaves as a non-specific abscess in any other part of the body—heals promptly and permanently. But when an abscess occurs so that either spontaneous or surgical drainage must be through the areola or at its edge, it has a remarkable tendency to recur repeatedly and to be highly resistant to permanent cure whatever the method of treatment. In one patient observed by the authors such a lesion flared up, drained pus and closed at intervals of a month or less for years. In other patients abscesses formed again after intervals of six months to more than two years of apparently complete quiescence. In a few instances the lesions remained as persistent draining sinuses.

The reason for this behavior is not entirely clear and may not, in fact, always be the same. The original infection may be in a gland of the skin or subcutaneous tissue but is by far most commonly in a duct of the breast. It has been suggested that lipids from the specialized apocrine glands of the areolar region, discharged into tissue by an incision, by spontaneous pointing or by a blow, may serve as a foreign body — in effect producing a chronically infected area of "fat necrosis." This is perhaps sometimes the explanation. However, the authors have not observed pathologic changes consistent with this theory in sections of the majority of the tissue specimens excised either from the walls of chronic abscesses or from the tissue surrounding them. The suggestion occurs that, if the infection is in a milk duct, the presence of a dilated ampulla with narrow duct mouth may mechanically form a "lake" in which chronic infection persists. This is in agreement with the pathological concept advanced by Zuska, Crile and Ayres in their explanation of persistent fistulas in this region. Confirmation of this theory is offered by the satisfactory results frequently obtained by complete excision of such an ampulla and is further suggested by observation of occasional cases in which indolent flare-up is relieved by drainage through the nipple.

Whatever the explanation, many recurring areolar abscesses eventually require more radical treatment than simple incision and drainage, and even after wide excision are prone to recur.

The authors have observed 64 patients with acute inflammatory lesions under the nipple and areola. In seven of the patients the lesions were bilateral, either at the same or separate times.

In a number of patients the initial lesion was a discrete tender lump with reddening of the skin, which subsided spontaneously without drainage, but in about half of the cases there was recurrence with eventual pointing and spontaneous or surgical drainage in most instances.

In three-fourths of the cases there were from one or two to literally dozens of recurrences of abscess formation. Recurrences were at irregular intervals. One patient had recurrence about every two weeks until mastectomy was done. In other cases quiescent intervals lasted several months to a year and occa-

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sionally several years. A few patients noted that recurrence came just before each menstruation. Two or three had recurrence during pregnancy or lactation. On the other hand, one patient who had had repeated recurrences had none during pregnancy, then abscess developed again after delivery.

ANTIBIOTICS

Neither the sulfa drugs nor the more recent galaxy of antibiotics have seemed to be very effective in either aborting or preventing recurrences. Organisms cultured when pus was available from a freshly opened abscess were usually staphylococcus aureus, hemolytic or non-hemolytic, or rarely streptococcus. In two cases observed within the past year an anaerobic organism was cultured from the pus. In one case the organism was sensitive to chloramphenicol and in the other to penicillin. The experience of the authors with treatment guided by antibiotic sensitivity tests has been too recent and too limited to permit conclusions. In no case was the content of an antibiotic in the blood maintained at an effective level for months at a time. Tubercle bacilli were not observed in smears, cultures or sections of tissue in any case.

Autogenous vaccines were prepared and used in treatment of some of the patients observed early in the present series, but apparently without benefit.

Irradiation at the time of the approach of recurrence or in the interval between abscess formations seemed equally ineffective.

SURGICAL TREATMENT

After it was realized that simple incision and drainage seldom effected cure, excision during an interval between acute episodes was tried. In earlier cases the scar of a quiescent abscess was excised well into normal skin, fat and breast and the wound closed with or without a small drain. Recurrence followed in almost all cases.

Next, in a number of cases, excision in an interval was done and the wound was left sufficiently open to permit packing until healing by granulation occurred. In a few cases the first attempt was successful, but in many abscess recurred in the scar.

In these earlier attempts to cure by excision, the importance of a remaining infected duct was not appreciated. In more recent operations, before excision was completed, the small chronic cavity which can usually be found under the scar was opened and search was made with a fine probe for a duct connecting with the nipple surface. If such a duct was found, the nipple was split and the duct wall to its mouth completely excised. One skin stitch was placed at the base of the nipple to restore its contour and a small pack was left through the nipple as well as through the open or partly open wound at the areolar edge. The final scar on the nipple surface was nearly or quite invisible. In five of the thirteen cases in which this operation was done, abscess recurred.

Following is a summary comparing results of excision of abscess scar with and without a duct to its mouth:

	Total	*Cured*
Excision without duct	28	7
Excision with duct	13	8

(The figures represent numbers of operations—not patients. In several patients, two or even more excisions were carried out.)

In the cases in which resection was followed by recurrence and in those in which for one reason or another resection was not attempted, the final outcome was either continuation of flare-ups, persistence of a sinus, resort to simple mastectomy, or a gradual subsidence of the process. In 21 cases the infection appeared gradually to diminish in virulence: Intervals between recurrences became longer and longer with less reaction and pain, and finally recurrences stopped; or, in a few instances, a sinus persisted for from a few weeks to several years without acute recurring abscesses. Nine patients still had discharge from the lesions or were having mild flare-ups when last observed—in one instance twelve years after the original abscess.

MASTECTOMY

In nine cases, because of the presence of multiple sinuses or of scar contraction and deformity of nipple and breast or because the patient became discouraged after one or more resections, simple mastectomy was finally done. One patient who, despite two local resections, had had recurrences about every two weeks, elected to have mastectomy. The following year a similar process began at the areola of the remaining breast. The patient, quite unwilling to face another prolonged, uncertain and painful course, insisted upon mastectomy at once.

Cancer was not observed in any of the 64 patients, all but seven of whom have been followed from two to twenty years.

490 Post Street.

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Complications of Intestinal Intubation

With Report of Two Cases of Unusual Complication

IRVING L. LICHTENSTEIN, M.D., *Beverly Hills*

• Many complications may arise from the use of intubation in treatment of intestinal obstruction. They are readily subject to classification and can be prevented by proper precautions.

Two illustrative case reports of unusual complications are presented.

USE OF WANGENSTEEN suction, although a large factor in reducing the mortality rate in cases of intestinal obstruction, is not without hazard. Recognition of the causes of complications from intubation will help to obviate them. Knowledge of how to deal with them once they have occurred can greatly reduce the morbidity.

Berger and Achs[1] succinctly outlined the role of intubation in each of three major clinical classifications of intestinal obstruction: (1) paralytic ileus without mechanical cause; (2) mechanical obstruction early; and (3) ileus immediately after operation. They reiterated the oft-repeated warning against procrastination and the use of intubation in cases in which the blood supply to any area of the bowel is impeded.

The danger of intubation is not limited, however, to cases in which there is strangulation; many additional complications have been reported since the use of suction tubes was introduced. The following reports are examples of unusual complications arising from the utilization of long tubes for intestinal decompression.

REPORT OF A CASE

CASE 1.—A 53-year-old man was admitted to the hospital with chief complaint of abdominal pain and vomiting of two days' duration. The patient gave a history of intermittent abdominal distention and colicky pain over a period of many years.

Upon physical examination, the abdomen was observed to be diffusely distended. Numerous loops of small bowel were palpated and peristaltic movement was noted. Intestinal colic was pronounced. On rectal examination good sphincteric tone was noted. There were no palpable masses and no ballooning. Soft brown feces were present. In an x-ray film of the abdomen diffuse distention of the small bowel was noted. A typical "step-ladder" pattern with much intraluminal fluid was observed (Figure 1).

Wangensteen suction with a Miller-Abbott tube was applied and parenteral alimentation and antibiotic therapy were carried out. A diagnosis of mechanical obstruction of undetermined cause was made.

Celiotomy was performed on the day of admittance. A thick adhesive peritoneal band arose from the pelvis, "producing mechanical obstruction at a point about six inches from the terminal ileum." No other evidence of intra-abdominal disease was found. Incision of the adhesion resulted in immediate ballooning of the cecum. Antibiotics and Wangensteen suction were used postoperatively.

Three days later the distention was not much reduced and no flatus was passed by rectum. "Therapeutic" spinal anesthesia was induced, but with little effect. On x-ray examination with barium enema the following day no colonic lesion was noted. The Miller-Abbott tube had extended to the distal ileum (Figure 2). Temperature, pulse and respirations remained essentially normal. Flatus was passed and there was diminution of distention. Fluids were given by mouth and the suction tube was clamped.

After the patient had been on full diet for 48 hours and bowel movements were normal, intestinal suction was considered no longer necessary. An intern, fearing retrograde intussusception, felt it was best to remove the tube through the anus. The metal adaptor was detached and the tube was permitted to progress down the pharynx. Five days later, despite persistent normal bowel movements, there was no evidence of descent of the tube. In an x-ray film, the entire Miller-Abbott tube was observed to be tightly coiled in the ileum (Figure 3).

Although there was slight abdominal distention with some discomfort, bowel movements and the temperature and pulse

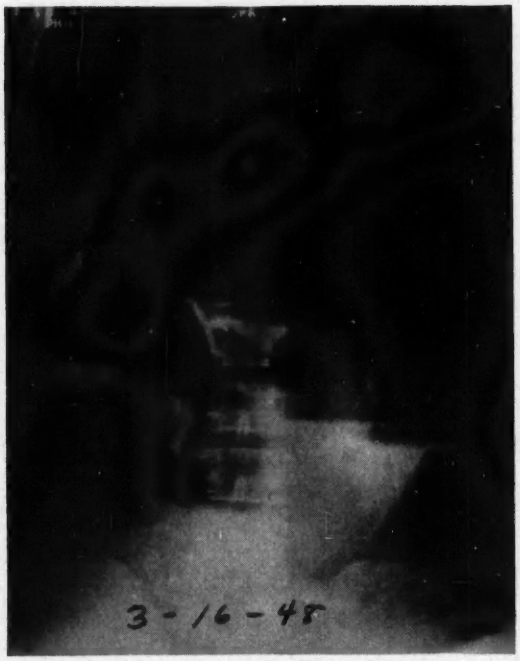

Figure 1.—Mechanical obstruction of small bowel.

and respiration rate continued to be normal. After x-ray study two days later, it was reported "the large loop of the tube appears to be open wider than on previous study. The head of the tubing appears to be extended further into the intestinal tract. It is possible that it is now uncoiling itself and starting through the intestines." (Figure 4.) The surgical opinion, however, was that the tube was tangled and that there was no evidence of progression. Accordingly, 13

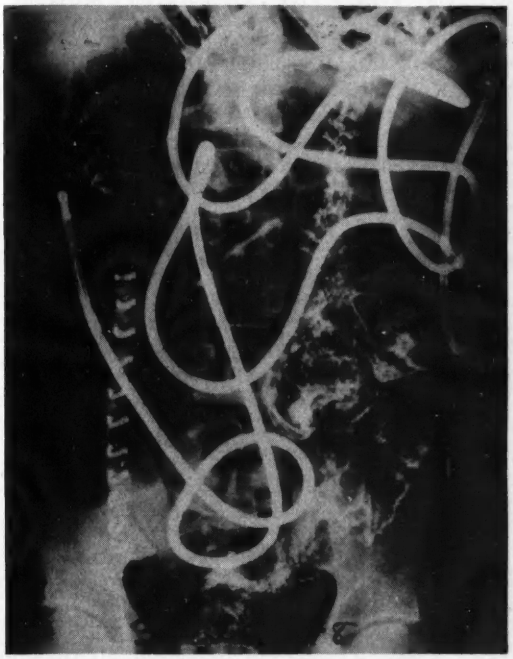

Figure 2.—Miller-Abbott tube progressing normally down the small intestines.

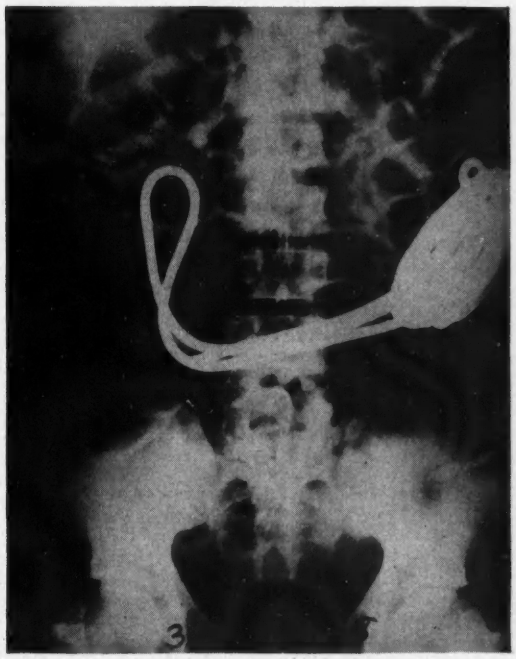

Figure 3.—Suction tube tightly coiled in ileum.

Figure 4.—Miller-Abbott tube remains knotted two days later.

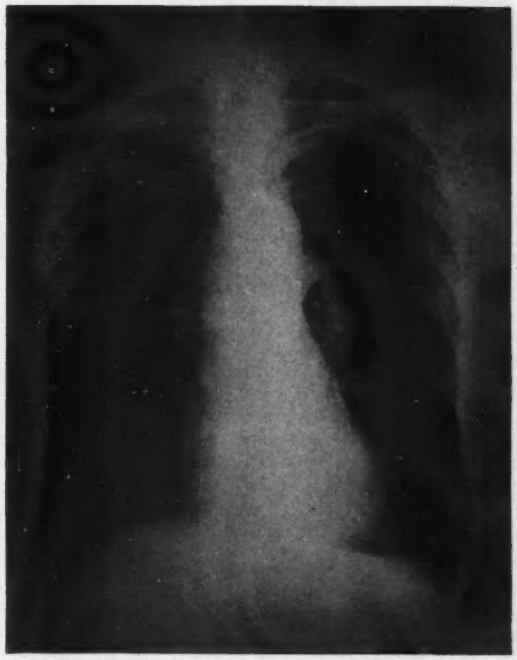

Figure 5.—Note the mercury particles, particularly in the right lower lobe. (Courtesy of J. Sugarman.)

days after the previous operation, the patient was operated upon again. A mass "approximately the size of an orange" was located 12 to 14 inches proximal to the ileocecal valve. The ileum at the site was incised on the anti-mesenteric surface, the coiled rubber tubing making up the mass was delivered in segments, and the bowel was closed.

Aside from a mild febrile reaction postoperatively, and moderate distention which responded to further intubation, the patient's subsequent course was uneventful. He was discharged from the hospital in good condition.

CASE 2.—Figure 5 is an x-ray film of the chest of a patient with intestinal obstruction (by permission of J. Sugarman). In this case, rupture of the mercury-filled intubation bag caused tracheal aspiration, with the majority of the mercury coming to rest in the right lower lobe. No serious residual effect was noted in four years of observation, however.

Certainly routine intestinal intubation, while not fraught with danger, has on occasion caused complications that made further treatment necessary. It would appear advantageous, therefore, to instruct members of the house staff in the management of intestinal tubes, and to make them cognizant of the many possible dangers attendant upon their use. A classified list of complications of intubation follows:

1. *Mechanical*
   - A. Balloon: (1) rupture, (2) over-distention.
   - B. Tube: (1) leaking, (2) kinking or knotting, (3) occlusion.
   - C. Adapters: (1) inaccurate fit, (2) errors due to labeling.

2. *Anatomical*
   - A. Respiratory Tract: (1) edema of turbinates, (2) epistaxis, (3) sinusitis, (4) eustachian inflammation, (5) otitis media, (6) laryngeal edema, (7) pneumonitis.
   - B. Gastrointestinal Tract: (1) pharyngitis, (2) esophageal erosion and stricture, (3) gastric erosion and perforation and hemorrhage, (4) intestinal erosion and perforation, (5) intussusception.

Rupture of the balloon with release of mercury or barium have both been reported without ill effect.[3, 7] Recently, too, the author observed a case of postoperative ileus in which mechanical obstruction was caused by overdistention of a Miller-Abbott tube bag with an excess of mercury. Similar complications caused by absorption of an excess of carbon dioxide by the bag within the intestinal tract have been reported.

The difficulties encountered with tubing may be kept to a minimum by discarding old tubes, and by frequent roentgenologic observation of the position of the tube in the patient. Brenizer[2] emphasized that tight coiling in storage may predispose to knotting. Occlusion of the tube by intestinal contents, a frequent complication, can be corrected by repeated irrigations.

Wyatt and Chisholm reported a case of intestinal obstruction caused by injection of Varco's solution into the wrong lumen. The patient died following a period of intestinal obstruction. They suggest the Honor tube in preference to the phalanged ends of the Miller-Abbott tube in order to avert such an error.

The respiratory complications of intubation are well known to surgeons. "Plugged nose" owing to swelling of the turbinates, epistaxis caused by erosion, and infection and edema owing to an encrusted tube's remaining too long in place, are not uncommon. Tracheotomy has been reported necessary in some instances.[4] In the main, these complications can be avoided by limiting the period of intubation, by occasionally injecting oil through the nostrils, and by inserting fresh tubes when intubation over long periods is necessary.

Vinson[6] reported esophageal stricture formation and Mahon[5] reported a case in which the tube caused gastric mucosal erosion and perforation with subsequent peritonitis. Prolonged presence of a Miller-Abbott tube in the small intestine has likewise been known to cause ulceration and perforation there.[1] Harris[3] reported intussusception accompanying overdistention of the balloon. The possibility of retrograde intussusception following too rapid withdrawal is well recognized by physicians who are familiar with the use of double lumen tubes. Frequent roentgenologic observation and avoidance of prolonged periods of intubation without change of tubes would appreciably decrease the number of such complications.

Prophylaxis is the best defense against complications. A resume of the salient prophylactic features follows: Check the balloon before insertion; use fresh tubing, not tightly coiled; flush the balloon with carbon dioxide; examine adapters and instruct hospital personnel; avoid prolonged intubation; insert oil in nostrils frequently; irrigate tube frequently; change tubing when used over extended periods; secure the proximal end of the tube unless the distal end has entered the cecum.

415 North Camden Drive.

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CASE REPORTS

- **Relapsing Fever Probably Caused by Borrelia Duttonii**
- **Anuria Owing to Urethral Obstruction by Sulfadiazine Crystals**
- **Aneurysm of the Abdominal Aorta with Rupture into the Duodenum**

Relapsing Fever Probably Caused by Borrelia Duttonii

MORRILL L. ILSLEY, M.D., *Claremont*

UP TO THE TIME of the case herein reported, the health officer for the southeast district of the Los Angeles County Health Department had had no reports of relapsing fever in the district since a physician in La Verne reported a case more than five years ago, although it is suspected that many persons had the disease in the interim and it was not recognized. The number of cases reported to the California State Department of Public Health in the five years 1945-49 averaged ten a year.[1]

REPORT OF A CASE

A white male college student 22 years of age had had intermittent chills and fever for 23 days when first observed September 24, 1951. He had been well until September 3, when, while at Big Bear Lake after twelve days on a camping trip, chills and fever developed suddenly and lasted a day and a night. At the same time the patient had headache, anorexia, vague abdominal pain, slight stiffness of the joints and a feeling of tightness in the chest. During the next week he felt well except for lassitude. Eight days after the onset, a second bout, which lasted three days, occurred. The temperature was 101 degrees F. and there was profuse sweating. Then again the patient was free of symptoms except for lassitude and loss of ten pounds in weight. Chills and fever recurred September 17 and lasted two days. A fourth attack, during which the patient vomited once, occurred September 24 and medical advice was sought.

Upon physical examination the patient appeared to be acutely ill. The tongue was coated. A few fine rales were heard in the hilar region of the chest. The abdomen was tympanitic, the spleen enlarged but not tender. In roentgen films the heart, lungs and mediastinum appeared to be normal.

The hemoglobin content of the blood was 15 gm. per 100 cc. and erythrocytes numbered 4,960,000 per cu. mm. The color index was 1.04. Leukocytes numbered 9,450—20 per cent lymphocytes, 8 per cent monocytes, 54 per cent segmented neutrophils, 17 per cent non-segmented neutrophils, and 1 per cent eosinophils. In bacteriological examination of the blood, no plasmodia were observed but many Borrelia spirochetes, probably Borrelia duttonii were present (Figure 1). Results of agglutination tests were negative for typhoid, paratyphoid and brucellosis. No organisms grew on a culture of the blood. The urine was not examined.

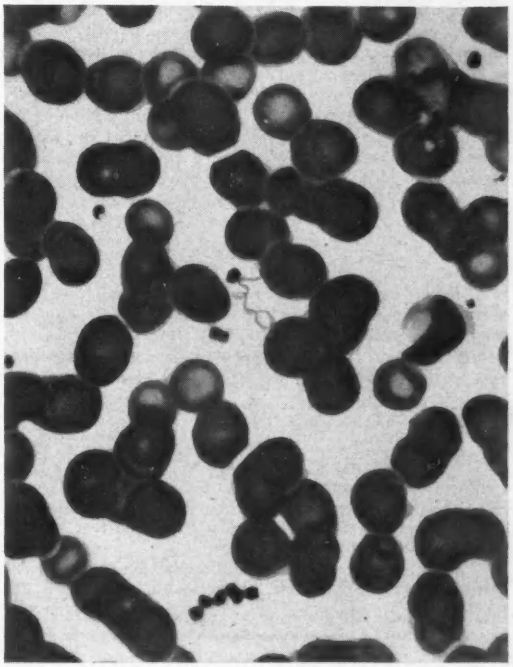

Figure 1.

Upon advice communicated from the clinic of Dr. Earle Moore at the Johns Hopkins University School of Medicine and Dr. T. B. Turner of the School of Hygiene and Public Health of the same university, the patient was given penicillin, 1,000,000 units daily for the first week, 600,000 units daily for the second week, and 300,000 units a day for the next five days. The fever abated soon after administration of penicillin was started, and the patient had no further attacks.

Textbooks mention relapsing fever caused by Borrelia duttonii, but few reports of cases could be found in the literature. Wynn and Beck[2] in a study of data on 283 cases that had been reported to the California State Department of Public Health between 1921 and 1944, made the following observations: In a majority of cases infection occurred along the Sierra Nevada range at or above 5,000 feet, where O. hermsi was the vector. In other cases the disease was con-

tracted at elevations between 250 and 4,500 feet, and in those cases O. parkerii was the vector. Rodent reservoirs (for example, chipmunks and tamarack squirrels) were proved in four cases—all over the 5,000-foot level. There was a seasonal pattern of occurrence of the disease. The peak months were June, July, August and September, a period following the emergence of rodents from hibernation, the opening of summer cabins, and influx of vacationists. However, there were some "out of season" cases in residents of endemic areas and it was conjectured that a disease colloquially called "squirrel fever" might be relapsing fever. The disease may be confused with brucellosis, malaria, pyelitis, influenza, typhoid, typhus, and sunstroke. It is characterized by sudden onset with fever, frontal headache, pain in the back, the limbs, and the joints, and pronounced prostration. Nausea and vomiting may occur. In a few instances in the California series, a rash was noted. The attacks usually last two to four days and the symptoms usually recur within three to twelve days. During the afebrile period, the patient feels entirely well. If no treatment is given, there may be as many as twelve relapses. In the majority of cases in the California series the patient had two to four attacks, a few as many as eight but each of decreasing severity. The infecting organism may be identified by either of two methods: microscopic examination of a smear of the blood, or animal inoculation. Smears should be prepared as thick or thin films of blood obtained at the peak of the attack before treatment is given. Wet preparations may be examined under the dark field if equipment is available.

SUMMARY

A patient who had several bouts of chills, fever with anorexia, headache and abdominal pain after a camping trip in a mountainous region of California was treated with penicillin after Borrelia spirochetes were noted in examination of the blood. The symptoms abated promptly and did not recur.

1111 Indian Hill Boulevard.

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Anuria Owing To Urethral Obstruction by Sulfadiazine Crystals

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ANURIA WITH OBSTRUCTION of the upper urinary tract has been reported on many occasions since the sulfonamide derivatives first came into general use.[1, 2-6] Anuria due to urethral obstruction secondary to sulfonamide therapy, however, while undoubtedly observed by other physicians, has not been as widely publicized. At the time the case herein reported was observed, the literature was reviewed and no report of anuria caused by crystalline obstruction of the urethra by a sulfonamide product was found. Later (in January, 1952) Dukes[2] cited four reports of cases in which lower urinary tract obstruction developed following treatment with sulfadiazine.

Presented before the Section on Pediatrics at the 81st Annual Session of the California Medical Association, Los Angeles, April 27-30, 1952.

REPORT OF A CASE

The patient, a boy aged 4 years and 8 months, was referred by another physician on February 7, 1951, because he had been completely unable to void that day after awakening in the morning. (Later it was determined that he had not voided for eighteen hours.) The referring physician, treating the patient for infection of the upper respiratory tract, had prescribed sulfadiazine and a total of 4 gm. of the drug had been taken at the time he was observed by the author. Upon physical examination, dullness upon percussion of the suprapubic region of the abdomen, suggestive of elevation of the dome of the bladder, was noted. There was pronounced stenosis of the external urethral meatus, and a whitish substance suggestive of crystals of sulfonamide derivative exuded from the orifice. First with a probe and later with a small sound, the external urethral meatus was dilated slightly and the packed crystals were broken to some extent. Thereafter, a small red rubber catheter was passed into the bladder and approximately 200 cc. of clear urine drained freely. In the course of catheterization, a considerable quantity of whitish crystalline material was expressed from the urethra. A specimen of the urine obtained from the bladder and subjected to routine analysis was noted to be within normal limits except that the urinary sediment contained many crystals of a structure typical of sulfonamide crystals. No organisms grew on a culture of the urine in 48 hours. After the catheterization, meatotomy was performed and the patient was sent home with instructions to force fluids and to return in five days for dilatation of the external urethral meatus. When the patient returned, the mother reported that he had voided freely after the initial therapy in the office, and had had no difficulty since that time.

DISCUSSION

The case reported illustrates one more complication to anticipate when prescribing sulfonamide therapy. Dukes said that one would expect this complication to occur more commonly in children than in adults because the diameter of the adult urethra is of sufficient size to permit the passage of the precipitated crystals, thus precluding obstruction. The patient in the case here reported had stenosis of the external urethral meatus, but whether this was a significant factor in causing urethral obstruction is unknown. However, it seems logical that any organic obstruction would tend to cause precipitation of the crystalline material and blockage of the urethral passageway. Dukes made no mention of stenosis of the external urethral meatus in the four patients that he reported upon.

The diagnosis was established easily in the present case because the patient was known to have been given sulfadiazine. Also, when the catheter was passed into the bladder, a satisfactory quantity of urine was obtained. This was at least enough to indicate that the patient had very little, if any, upper urinary tract obstruction. There did not appear to be any evidence of toxic nephrosis in the patient in the present case or in the patients reported upon by Dukes.

The treatment, namely stopping the drug, catheterization, meatotomy and forcing fluids by mouth, relieved the patient at once. Other measures, suggested by Dukes are alkalization of the urine and hot sitz baths. Dukes also mentioned that, if these measures fail, removal of the granular material by urethroscopic means under general anesthetic may become necessary.

The prognosis for complete recovery from this complication seems excellent. In all cases reported, the patients recovered promptly and without any known sequelae. Apparently the occurrence of this complication need not preclude further sulfonamide therapy. The patient in the present case

received comparable dosages of sulfadiazine on two occasions after recovery from the urethral obstruction, without urinary complications.

SUMMARY

A case history of a child with urethral obstruction and anuria due to sulfadiazine crystals is presented. Stenosis of the external urethral meatus which was present in this case may have contributed to the blockage by narrowing the urethral channel. Treatment which consisted of discontinuing the drug, catheterization, meatotomy and forcing fluids by mouth, resulted in prompt recovery.

300 Homer Avenue.

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Aneurysm of the Abdominal Aorta with Rupture into the Duodenum

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RUPTURE OF AN ANEURYSM into the gastrointestinal tract is rare. Hunt and Weller[2] who reported a case in 1946 found reports of only 40 cases in the literature. In 33 cases the rupture was into the duodenum. By 1951, 43 such cases had been reported.[1] The following is a report of an additional case.

REPORT OF A CASE

A 72-year-old white male one morning vomited a large amount of bright red blood after nausea and generalized abdominal distention of one week's duration. When examined about 15 minutes later the patient was in a state of shock. The systolic blood pressure was 110 mm. of mercury and diastolic pressure could not be measured. The patient refused hospitalization and was placed on a regimen of milk and Amphojel® every two hours. Tarry stools were passed. In the evening the blood pressure became stabilized at 130 mm. systolic and 80 mm. diastolic. During the night several large tarry stools were passed and the patient vomited a material having the appearance of coffee grounds. The next morning the patient collapsed in the bathroom and was taken to the hospital where he was admitted in a state of shock. Twenty years previously a diagnosis of peptic ulcer had been made following an episode of hematemesis. In the interim the patient had been in good health.

Upon physical examination, diffuse epigastric tenderness was noted, but there were no palpable abdominal masses or organs. The hemoglobin content of the blood was 10.5 gm. per 100 cc. Erythrocytes numbered 3.2 million per cu. mm. and leukocytes were 14,250—92 per cent polymorphonuclears, 6 per cent lymphocytes and 2 per cent monocytes.

Hematemesis and melena continued, necessitating four transfusions. Three days after admittance to the hospital the patient had sudden pain in the left side of the chest. Dyspnea and cyanosis developed and the patient died.

At autopsy the heart was observed to be of normal size; it weighed 350 gm. There was only minimal arteriosclerosis of the coronary arteries. A few pinpoint sized atheromatous plaques were present in the ascending aorta. There was moderate atherosclerosis of the abdominal aorta, with a saccular aneurysm containing a laminated thrombus situated just above the bifurcation. The aneurysmal sac was 3 cm. in diameter and 6 cm. in length and projected 3 cm. anteriorly. A probe was readily passed from the site of rupture in the anterior wall of the sac to a perforation in the duodenum (Figure 1). The perforation, which was 1 cm. in diameter, lay on the posterior aspect of the third portion of the duodenum, 2 cm. distal to the papilla of Vater (Figure 2). The stomach contained about 1,500 cc. of blood and clots. The

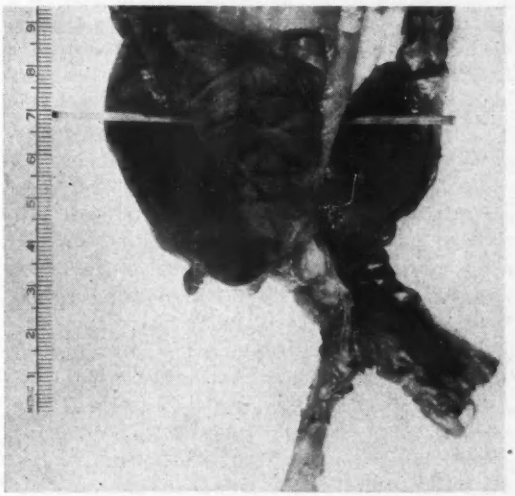

Figure 1.—A probe was passed from the site of rupture in the anterior wall of the sac to a perforation in the duodenum.

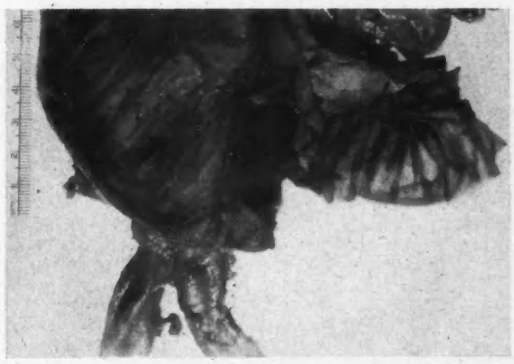

Figure 2.—Arrow points to perforation of the duo denum 2 cm. distal to the papilla of Vater.

proximal small bowel was filled with clotted blood. The colon contained tarry material. There was slight erosion of the anterior surfaces of the bodies of the third and fourth lumbar vertebrae.

In microscopic examination of sections taken at the level of the midthoracic aorta, intimal atheromatous deposits and atrophy of the media were observed. At the site of rupture, there was extensive hyalin change with loss of the smooth muscle and elastic tissue of the media; and a hyalinized thrombus was attached to the intima.

DISCUSSION

A correct clinical diagnosis was not made in the case here reported. The history of previous hematemesis due to peptic ulcer and the absence of a palpable abdominal mass indicated a clinical diagnosis of peptic ulcer. The terminal symptoms suggested coronary occlusion, but this diagnosis was not confirmed at autopsy.

With the inclusion of this case, there are 44 cases of abdominal aneurysm with rupture into the duodenum reported in the literature. In a previous report of eight cases observed in 16,633 autopsies,[1] it was noted that the lesion occurred in elderly persons; the average age in the eight cases was 72 years. Arteriosclerosis with or without hypertension was considered to be the most important cause of the lesion.

Rupture into the duodenum appears to be purely fortuitous, dependent on the proximity and fixation of the second and third portions of the duodenum to the lower abdominal aorta. Symptoms are usually of short duration, and pain is the usual initial manifestation. Melena and especially hematemesis are grave prognostic signs, suggesting a rapidly fatal outcome. A pulsating abdominal mass is usually present. Expansile pulsations are said to be highly suggestive of aneurysm,[4] and may be associated with a thrill or bruit over the mass. X-ray studies are frequently helpful in diagnosis.[3] Treatment is unsatisfactory and the prognosis is poor.

3024 Pine Street.

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California MEDICINE

EDITORIAL

PR—One Year Later

THE DESIGNATION "PR" has come more and more into medical usage in the past few years, to denote the public relations of the profession. Throughout the country, PR is becoming widely used and recognized, not as a symbol but as a program of the profession in achieving and publicizing its good deeds and eliminating its less fortunate incidents.

State medical journals, county society bulletins and national publications of the A.M.A. and other nationwide organizations are devoting important space to the programs and accomplishments of local, state and national groups in achieving "good performance, properly understood and adequately publicized."

The California Medical Association has long recognized the need of a high degree of performance in the field of public relations. Political and economic pressures, often based on misinformation, false premises or frankly political self-seeking have made the association extraordinarily alert to defend the position of medicine in this state by eliminating all possible causes of complaint by patients and by maintaining medical performance of the highest character. Only when these two functions are fully performed has medicine something to tell the public.

Just one year ago the C.M.A. set up its own public relations department. Staffed by a director and two associates, located in both northern and southern offices, this department has turned in its report for its first year of existence. The report makes excellent reading.

California's PR program was based initially on achieving two basic guarantees to the public. The first was the guarantee of the availability of the services of a doctor of medicine under any and all predictable circumstances. The second was the guarantee of recourse against real or fancied abuses of patients by physicians. Both guarantees revolve around proper organization of the county societies, a form of organization which permits the sharing of burdens which may be placed upon physicians under the guarantee of services and which demands of society members an objective attitude in seeing to it that a complaining patient is given a square deal and a chance to be heard.

From this starting point, and with the caution in mind that the best progress is made by taking one step at a time, the public relations department has concentrated its activities toward perfecting, through the county societies, the methods of carrying out a few basic responsibilities which the public puts upon medicine. It has undertaken to explain to the public the distinctions between a doctor of medicine and the various other kinds of practitioners who deal with sick persons. It has helped to organize, and to publicize, the availability of physicians at all hours through well-planned medical society emergency telephone services. And it has arranged for the broadcast of radio programs which give further

CALIFORNIA MEDICINE

OWNED AND PUBLISHED BY THE CALIFORNIA MEDICAL ASSOCIATION

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For information on preparation of manuscript, see advertising page 7

evidence of the high quality of training and service of California doctors of medicine.

Newspaper advertising copy headed "Your M.D." has appeared over the name of the appropriate county medical society in 716 newspapers circulated in all counties in California. These societies serve about 82 per cent of the total population of California.

Radio programs, each consisting of a 13-week series, have been scheduled on 91 radio stations in California as public service programs. The stations cover practically all of the state's population.

Emergency telephone service is now established in 31 of the 40 county societies of the state, representing better than 93 per cent of the C.M.A. membership and a like percentage of the general population.

These accomplishments, made possible only through the active work and cooperation of the county societies, are easily listed as statistics. Much more difficult is the task of making them work and of ironing out the many rough spots encountered in every instance. The department's staff has found its job to be at least a full-time chore. Travel, meetings, conferences and the insistent demand for perfection in detail have made extraordinary demands on the time and talents of the staff members. To their credit, they have discharged their obligations with an unusually high sense of integrity and with great capability.

The true measure of accomplishment in this, a field in which results admittedly are intangible, is the general response on the part of publishers, radio and television operators and others whose daily business is to report to the public and, directly or indirectly, to mould public opinion. By this measure, the C.M.A. performance gives every indication of meriting high praise. A recent example came in the case of a newspaper publisher who was happy to receive the true facts and to report them to his readers after a young man had secured considerable notoriety by offering to sell his eyes to secure funds to care for his mother, who had cancer. Through a member of the C.M.A. staff, the county society and a hospital, assurance was given the publisher that the mother could obtain needed care, regardless of her financial position, and the publisher was quick to give his readers this fact.

The pleasure of reporting good accomplishments is often mitigated by the realization that much more remains to be done. Thus, in honesty, the C.M.A. public relations department recognizes that its job is but fairly begun. Much remains to be done and many items will be added to the program, in orderly fashion, in the coming years. However, much groundwork has been done which will need little or no repetition. A higher sense of responsibility than they had before has been instilled into some county societies and some members. A good portion of the public has been favorably impressed with the desire of the medical profession to do a good job, professionally, ethically and economically. With this background, we can look to the future with more optimism than we have had in the past and with confidence in attaining the goal of "good performance, properly understood and adequately publicized."

LETTERS to the Editor . . .

Psychosomatic Kidney Stones

Butt and Hauser[1] of Butt-Douglas Medical Foundation, Pensacola, Florida, report detailed physiochemical studies of the urines of 680 individuals of different ethnic groups in Florida and islands of the Pacific. They found a correlation between the presence or absence of hydrophilic colloids in these urines and the tendency of patients to develop kidney stones. In the absence of such colloids kidney stones are relatively frequent, while stones rarely form in the presence of protective urinary colloids.

Therapeutic test of this conclusion was made on a patient with multiple bilateral rapidly forming recurrent kidney stones. The urine of this patient showed numerous microscopic crystals. One hour after subcutaneous injection of 300 TR-units of hyaluronidase, his urine became free from crystals. Hyaluronidase therapy was therefore instituted in twenty similar cases and continued for a period of eleven to fifteen months. During this time eighteen of these patients remained free from recurrent urinary calculi.

As a collateral observation Butt and Hauser noted that "the formation of protective colloids in the human body virtually disappears during times of strong emotional stress," a conceivable basis for some future psychosomatic theory of kidney stones.

W. H. MANWARING, M.D.
Palo Alto

REFERENCE

1. Butt, A. J., and Hauser, E. A.: The importance of protective urinary colloids in the prevention and treatment of kidney stones, Science, 115:308, Mar. 21, 1952.

California MEDICAL ASSOCIATION

NOTICES & REPORTS

Executive Committee Minutes

Tentative Draft: Minutes of the 233rd Meeting of the Executive Committee of the California Medical Association, San Francisco, August 10, 1952.

The meeting was called to order by Chairman Lum in the Association offices, 450 Sutter Street, San Francisco, at 10:00 a.m., Sunday, August 10, 1952.

Roll Call:

Present were President Alesen, President-Elect Green, Council Chairman Shipman, Speaker Charnock, Auditing Committee Chairman Lum and Editor Wilbur. Absent for cause, Secretary Daniels.

Present by invitation during all or part of the meeting were Dr. Dwight H. Murray, legislative chairman; Dr. Francis J. Cox, chairman of the Committee on Industrial Accident Commission; Dr. Lewis T. Bullock, chairman of the Committee on Psychology; Dr. Sam Sherman, representing the San Francisco Medical Society; Frank G. Dickinson, Ph.D., director of the Bureau of Medical Economic Research of the American Medical Association; Mr. Ben Read of the Public Health League of California, and Messrs. John Hunton, Ed Clancy and Robert L. Thomas of C.M.A. staff.

1. *American Medical Education Foundation:*

Discussion was held on the organization of members to solicit contributions to the American Medical Education Foundation. On motion duly made and seconded, it was voted to handle the solicitation through the county societies and the members of the Council and to appoint Dr. John W. Green chairman of a steering committee. Other members of this committee were selected as Councilors Bostick, Wheeler, Dau and West.

2. *Diagnostic Devices:*

The question of non-medical personnel using diagnostic devices was discussed and on motion duly made and seconded, it was voted to appoint a committee of the Council to consider this problem. Dr. J. Philip Sampson was named as chairman and Councilors Morrison and Ray as members.

3. *County Society Secretaries:*

On motion duly made and seconded, it was voted to appoint Councilor Morrison as chairman and Drs. A. E. Moore of San Diego and J. E. Young of Fresno as members of a committee to consider means of expressing appreciation to secretaries of the county societies for their valuable services.

4. *Woman's Auxiliary:*

On motion duly made and seconded, it was voted to appoint Drs. Sam J. McClendon and R. Stanley Kneeshaw as members of the advisory committee to the Woman's Auxiliary, to serve with the president, president-elect and secretary of the Association.

5. *A.M.A. Public Relations Institute:*

On motion duly made and seconded, it was voted to limit the Association's representation at the September public relations institute of the American Medical Association to the executive secretary and two public relations department representatives.

6. *State Department of Public Health:*

(a) On motion duly made and seconded, it was voted to refer to the Committee on Public Health and Public Agencies an invitation from the State Department of Public Health to attend a conference on the problems of prematurity.

(b) Discussion was held on a conference planned for August 12 in Santa Barbara on the Crippled Children's Act. Dr. Harry E. Henderson and Mr. Ben Read were to attend the conference as Association representatives. On motion duly made and seconded, it was voted to meet with representatives of other organizations on similar problems.

7. *Public Policy and Legislation:*

(a) On motion duly made and seconded, it was voted to appoint Dr. Cullen Ward Irish as chairman and Drs. John B. Doyle, Edwin E. McNiel, Abram E. Bennett and Paul A. Gliebe as a committee to cooperate with state officials on problems affecting medical care. This committee may be assisted by advisory members to be selected by the Council.

(b) A complaint from a group of practical nurses on the regulations adopted by the Board of Vocational Nurse Examiners was considered and it was agreed to refer this to the committee appointed for this purpose, Dr. Howard Naffziger, chairman.

8. *Medical Services Commission:*

Dr. Shipman reviewed the meeting of the executive committee of the Medical Services Commission, which has scheduled its organization meeting for August 23-24. Dr. Sam Sherman and Frank G. Dickinson, Ph.D., discussed some of the problems to be presented to this commission.

9. *Committee on Psychology:*

Dr. Lewis T. Bullock reviewed the meetings held by his committee with representatives of other organizations and presented the draft of proposed legislation to provide for licensing clinical psychologists. It was agreed to invite Dr. Bullock to present this matter to the next meeting of the Council.

10. *President's Health Commission:*

Report was made that the President's Health Commission has scheduled a series of public hearings, one of which will be held in San Francisco on September 29, 1952. It was agreed that the Association should request permission for a representative to appear before this hearing.

11. *Los Angeles County Medical Association:*

A request from the Los Angeles County Medical Association for appointment of a referee to hear charges of unprofessional conduct brought against one of its members was received and, on motion duly made and seconded, it was voted to comply with this request, legal counsel to select the referee.

Adjournment:

There being no further business to come before it, the meeting was adjourned at 5 p.m.

DONALD D. LUM, M.D., *Chairman*
SIDNEY J. SHIPMAN, M.D., *Acting Secretary*

In Memoriam

CLEMENT, THOMAS G. Died in Healdsburg, March 26, 1952, aged 65, of coronary occlusion. Graduate of the University of Colorado School of Medicine, Denver, 1910. Licensed in California in 1947. Doctor Clement was a member of the Sonoma County Medical Society, the California Medical Association, and the American Medical Association.

✤

COLLINGS, CLYDE W. Died in Encino, July 4, 1952, aged 60, of coronary artery disease. Graduate of the University of Oregon Medical School, Portland, 1919. Licensed in California in 1939. Doctor Collings was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

✤

GRAHAM, HARRINGTON B. Died in San Francisco, July 20, 1952, aged 78. Graduate of the University of California Medical School, Berkeley-San Francisco, 1899. Licensed in California in 1899. Doctor Graham was a retired member of the San Francisco Medical Society, and the California Medical Association.

✤

HARRIS, EVA L. Died in San Francisco, July 9, 1952, aged 84. Graduate of Hahnemann Medical College of the Pacific, San Francisco, 1895. Licensed in California in 1895. Doctor Harris was a retired member of the Alameda-Contra Costa Medical Association, and the California Medical Association.

✤

KARSHNER, ROLLA. Died in Los Angeles, July 12, 1952, aged 64. Graduate of the University of Michigan Medical School, Ann Arbor, 1917. Licensed in California in 1919. Doctor Karshner was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

✤

MUSSER, FRED C. Died in Los Angeles, July 1, 1952, aged 64, of coronary artery disease. Graduate of Wayne University College of Medicine, Detroit, 1915. Licensed in California in 1943. Doctor Musser was a member of the Los Angeles County Medical Association, the California Medical Association, and the American Medical Association.

✤

WILSON, JOHN W. Died in San Francisco, July 14, 1952, aged 86. Graduate of the State University of Iowa College of Homeopathic Medicine, Iowa City, 1894. Licensed in California 1895. Doctor Wilson was a retired member of the San Francisco Medical Society, and the California Medical Association.

CALIFORNIA MEDICAL ASSOCIATION

Annual Meeting

LOS ANGELES
May 24-27, 1953

Papers for Presentation

If you have a paper that you would like to have considered for presentation, it should be submitted to the appropriate section secretary (see list on this page) not later than November 15, 1952.

Scientific Exhibits

The space available for scientific exhibits is limited. If you would like to apply for space, please write immediately to the office of the California Medical Association, 450 Sutter Street, San Francisco 8, for application forms. To be given consideration by the Committee on Scientific Work, the forms, completely filled out, must be in the office of the California Medical Association not later than December 1, 1952. (No exhibit shown in 1952, and no individual who had an exhibit at the 1952 session, will be eligible until 1954.)

SCIENTIFIC PAPERS . . .

. . . SCIENTIFIC EXHIBITS

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AN EARLY START HELPS

SECRETARIES OF SCIENTIFIC SECTIONS

Allergy . . . . . *Norman Shure*
6317 Wilshire Boulevard, Los Angeles 48

Anesthesiology . . . . . *Joseph H. Failing*
475 Buena Vista Street, San Marino 9

Dermatology and Syphilology . . . *Frances Keddie*
300 Homer Avenue, Palo Alto

Eye, Ear, Nose and Throat—

Eye . . . . . *Alfred R. Robbins*
1930 Wilshire Boulevard, Los Angeles 5

ENT . . . . . *Francis A. Sooy (Asst. Sec.)*
490 Post Street, San Francisco 2

General Medicine . . . . . *William D. Evans*
10655 Riverside Drive, North Hollywood

General Practice . . . . . *A. Bradford Carson*
1515 Fruitvale Avenue, Oakland 1

General Surgery . . . . . *Arthur C. Pattison*
960 East Green Street, Pasadena

Industrial Medicine and Surgery . *Dan Kilroy (Asst. Sec.)*
3300 Third Avenue, Sacramento

Obstetrics and Gynecology . . . *Harold K. Marshall*
229 North Central Avenue, Glendale 3

Pathology and Bacteriology . . . . *A. R. Camero*
679 South Westlake Avenue, Los Angeles 5

Pediatrics . . . . . *Clement J. Molony*
416 North Bedford Drive, Beverly Hills

Psychiatry and Neurology . . . . . *A. E. Bennett*
2000 Dwight Way, Berkeley 4

Public Health . . . . . *Charles E. Smith*
U. C. School of Public Health, Berkeley 4

Radiology . . . . . *Calvin L. Stewart*
2330 First Avenue, San Diego 1

Urology . . . . . *James A. May*
2001 Fourth Avenue, San Diego 1

Q. & A. on C.P.S.

Question: **What information should be obtained for a doctor's office records from a patient who presents a C.P.S. identification card?**

Answer: Obtain the patient's full name, address, age, relationship (indicate whether a subscribing member or family member, because they may receive different benefits), C.P.S. group and member number, and ask patient to sign and verify income status. Other necessary and pertinent data are obtained from the contract code numbers appearing on the member's C.P.S. identification card.

Question: **What steps should a C.P.S. physician follow when billing for services rendered for a procedure not specifically listed in the C.P.S. Fee Schedule?**

Answer: When billing for an unlisted procedure, a written report of explanation should accompany the billing form and be addressed to the attention of the Medical Policy Committee. If this prescribed method is followed by the C.P.S. physician's office, the billing and report of explanation submitted will be especially handled for appropriate consideration to establish a fee comparable to a fee schedule item of closest similarity.

Question: **Will C.P.S. make payments for services rendered by two physicians who are in attendance on the same case at the same time?**

Answer: Only where it is warranted by the necessity of supplementary skills in cases of unusual complications or severity. Under such circumstances a written report of explanation will eliminate the need of further correspondence relating to the case. In this manner prompt payments will be assured.

Question: **I have been requested several times to submit medical reports each month on my veteran patients. Is this reporting necessary?**

Answer: An adequate medical report is necessary each month for several reasons. A veteran is eligible for treatment at government expense only for the condition or conditions for which he has been rated as service-connected. Adequate medical reporting is essential so that the authorizing official of the Veterans Administration can be sure that government funds are being spent in a legal manner. Conditions receiving treatment for which the veteran is not service-connected should be paid for by the veteran as a private patient. Physical findings are also important in order that the severity of the veteran's present condition may be evaluated by the Veterans Administration. These findings have a definite bearing on the amount and type of treatment which can be authorized. They also have a direct bearing on whether conditions adjunct to a service-connected condition will be covered at government expense.

Question: **Are Korean veterans eligible for out-patient treatment under the Veterans Home Town Care Program?**

Answer: A new directive received by C.P.S.-VA is designed to cut red tape and grant immediate out-patient medical care to Korean War veterans. One year (or more in cases of psychosis, tuberculosis, multiple sclerosis, etc.), immediately following the veteran's discharge, is designated as a "presumptive period" during which any medical disability arising is "deemed to have been the result" of the veteran's period of active service in the Armed Forces.

Immediately upon receipt of a claim from the veteran (Form 526), authority for treatment is issued on a prima facie basis to the physician of the veteran's choice, without waiting for approval of such claim by the Adjudication Boards.

Question: **Why does C.P.S. require certain members to complete endorsements of service for pre-existing conditions, while in other cases this is not required?**

Answer: Any member who applies for *group* membership when first eligible is not required to fill out these forms. However, underwriting experience clearly demonstrates that those persons who are "self-selected risks" (a person who did not enroll in C.P.S. at the time a group was first formed) should only be accepted subject to a statement of health, and exclusions of benefits for all preexisting conditions. This policy is sound from an underwriting standpoint, and serves the best interests of all C.P.S. members. In this manner, those members enrolling in C.P.S. when first eligible, who in most cases have no knowledge of their immediate need for services, will receive maximum protection.

Question: **When a C.P.S. member leaves his place of employment and transfers to the Direct Payment Program, where the Two-Visit-Deductible coverage is no longer available, is the Medical-Care-While-Hospitalized contract considered a new contract?**

Answer: If a member had Two-Visit-Deductible coverage under his group contract, and received maximum benefits under that contract for any condition, he would still be eligible for completely new benefits for the same condition, if he should take out Medical-Care-While-Hospitalized on the Direct Payment contract.

NEWS & NOTES

NATIONAL · STATE · COUNTY

LOS ANGELES

Dr. L. A. Alesen, president of the California Medical Association, will act as moderator in a panel discussion on "The Physician's Public Relations" at the seventeenth alumni **postgraduate convention** of the Alumni Association of the College of Medical Evangelists Medical School, to be held in Los Angeles, March 8 to 13, 1953.

Dr. George W. Stephenson, assistant director of the American College of Surgeons, and specialists from the Johns Hopkins University Medical School, the Indiana University Medical School, and the Lahey Clinic in Boston are among other speakers. Designed for general practitioners, the meeting will include a varied program of lectures, panels, luncheon round-tables, refresher courses, exhibits and women's activities.

* * *

The 1952 **Scientific Assembly** of the California Academy of General Practice will be held November 9 to 12 at the Statler Hotel, Los Angeles.

* * *

Six outstanding guest speakers will participate in a two-day program of **symposia on pharmacology, hypertension and peripheral vascular disease** which is to be held October 15 and 16 at the Wilshire-Ebell Theatre under the sponsorship of the Los Angeles Heart Association. They are C. Sidney Burwell, M.D., research professor of clinical medicine, Harvard Medical School; Julius H. Comroe, Jr., M.D., professor of physiology and pharmacology, University of Pennsylvania Graduate School of Medicine, and professor of clinical physiology, University of Pennsylvania; Keith S. Grimson, M.D., professor of surgery, Duke University; Robert L. King, M.D., associate clinical professor of medicine, University of Washington School of Medicine; Harry Mandelbaum, M.D., associate in medicine, New York State University, College of Medicine; and M. L. Tainter, M.D., formerly professor of pharmacology, Stanford University, and now trustee of the Albany College of Pharmacy.

The program for the meeting follows:

WEDNESDAY MORNING, OCTOBER 15

SYMPOSIUM ON PHARMACOLOGY

In Memory of Morris H. Nathanson, M.D.

Gordon A. Alles, Ph.D., Moderator

9:30—Oral Diuretics in the Treatment of Congestive Failure—Sim P. Dimitroff, M.D., and M.C. Thorner, M.D.

9:45—Usage of Digitalis Glucosides—William Paul Thompson, M.D.

10:15—Physiopharmacologic Action of the Sympathomimetic Drugs, Maurice L. Tainter, M.D.

10:45—Physiopharmacologic Action of the Antihypertensive Drugs—Julius H. Comroe, M.D.

11:15—Use of Isotopes in Cardiovascular Disease States:

1. The Detection of Thyrotoxic Heart Disease—Franz K. Bauer, M.D.
2. The Treatment of Angina Pectoris and Congestive Heart Failure with Radioactive Iodine—Henry L. Jaffe, M.D., Maurice H. Rosenfeld, M.D., Frederick W. Pobirs, M.D., and Lawrence J. Stuppy, M.D.

Wednesday Afternoon

SYMPOSIUM ON HYPERTENSION

Harry Goldblatt, M.D., Moderator

1:30—The Natural History of Hypertension — C. Sidney Burwell, M.D.

2:15—Diagnostic Procedures in the Selection of Patients for Definitive Treatment of Arterial Hypertension — Keith S. Grimson, M.D.

2:35—The Role of the Adrenal in Essential Hypertension with an Evaluation of Adrenalectomy—Julius H. Comroe, M.D.

3:20—The Management of Essential Hypertension by Medical and Surgical Means—Keith S. Grimson, M.D.

4:05—Round Table on Hypertension—George C. Griffith, M.D., chairman; C. Sidney Burwell, M.D., Keith S. Grimson, M.D., Julius H. Comroe, M.D., Robert L. King, M.D., Maurice L. Tainter, M.D., and Harry Goldblatt, M.D.

THURSDAY MORNING, OCTOBER 16

SYMPOSIUM ON PROCEDURE

In Memory of Eugene B. Levine, M.D.

Edward Phillips, M.D., Moderator

9:30—The Clinical Value of Vectorcardiography—Stephen R. Elek, M.D., and B. J. Allenstein, M.D.

9:50—The Clinical Significance of Ballistocardiography—Harry Mandelbaum, M.D.

10:20—The Value of Cardiac Catheterization and Angiocardiography in Congenital Heart Disease—Sidney S. Sobin, M.D., Charles R. Baker, M.D., John L. Johnson, M.D., E. Levin, M.D., and Walter S. Thompson, M.D.

10:40—A Five-Year Study of Increasing Bacterial Resistance to the Antibiotics in Subacute Bacterial Endocarditis—David C. Levinson, M.D.

11:00—The Physiologic Aspects and Management of Heart Disease in Pregnancy—C. Sidney Burwell, M.D.

Thursday Afternoon

SYMPOSIUM ON PERIPHERAL VASCULAR DISEASE

William D. Evans, M.D., Moderator

1:30—The Clinical Evaluation of the Patient with Obliterative Arterial Disease of the Extremities—Ellen Brown, M.D.

2:00—The Place of Sympathectomy in the Treatment of Occlusive Arterial Disease of the Extremities—Lawrance N. Atlas, M.D.

2:30—Surgery of Large Arteries in Atherosclerotic Vascular Disease—Norman E. Freeman, M.D.

3:15—Emergency Treatment of Acute Vascular Occlusions:

1. Medical—Julius H. Comroe, M.D.
2. Surgical—C. J. Berne, M.D.

4:05—Round Table on Peripheral Vascular Disease—Travis Winsor, M.D., chairman; Ellen Brown, M.D., Lawrance N. Atlas, M.D., Norman E. Freeman, M.D., Julius H. Comroe, M.D., C. J. Berne, M.D., C. Sidney Burwell, M.D., and Robert L. King, M.D.

Two medical schools in Los Angeles have been granted funds by the National Cancer Institute **to aid studies on cancer control.** The University of California School of Medicine received $12,045 for a study of the ecology of cancer of the uterine cervix by Drs. Edward G. Jones and Ian Macdonald, and a grant of $10,000 was made to the University of California at Los Angeles for a program to develop better methods of cancer teaching in nursing schools which is to be supervised by Lulu K. Wolf, R.N.

MENDOCINO

Dr. Neely E. Bradford, formerly of Sonoma, recently was appointed medical director of the Mendocino County Hospital at Ukiah. He succeeds Dr. H. O. Cleland.

MONTEREY

Dr. Charles S. Herbert, chief of chest services at the Veterans Administration Hospital at Albuquerque, N. M., has been appointed medical superintendent of Monterey County Hospital. Dr. Herbert is to take over his new duties October 1. The hospital has been without a medical superintendent since Dr. John C. Sharp resigned the post last January.

SAN DIEGO

A symposium on heart disease, sponsored by the San Diego County Heart Association, will be held October 13 in the auditorium of the U. S. Naval Hospital in Balboa Park. The program will begin at 9:30 a.m. with an address by Dr. Lewis Bullock, president of the California Heart Association. Other speakers and their subjects: Dr. William Paul Thompson, Los Angeles, "Usage of Digitalis Glucosides"; Dr. C. Sidney Burwell, research professor of clinical medicine of Harvard Medical School, "The Physiological Aspects of Management of Heart Disease in Pregnancy"; Dr. Julius H. Comroe, Jr., professor of physiology and pharmacology, University of Pennsylvania Graduate School of Medicine, "Mode of Action of Drugs Currently Used in the Treatment of Hypertension"; Dr. Keith S. Grimson, professor of surgery, Duke University, "Surgical Management of Hypertension."

Dr. Burwell will also be the speaker at a dinner meeting to be held in the Manor Hotel, San Diego, beginning at 7:30 p.m.

SAN FRANCISCO

The **Florence Crittenton Home** of San Francisco, since 1889 a resource to physicians called upon to counsel unmarried expectant mothers regarding their problems and care, has just published a leaflet outlining its services and answering questions most frequently asked about the home's programs. The San Francisco home primarily serves women from Northern California communities, while a Crittenton Home at 234 East Avenue 33, Los Angeles, provides similar services in the Southern California area.

Any physician who wishes to become better acquainted with the San Francisco home may obtain a copy of the new leaflet by writing to the home, at 850 Broderick Street, San Francisco.

* * *

The San Francisco Heart Association's twenty-third annual **postgraduate symposium on heart disease** will be held October 22 to 24 in the Colonial Ballroom, St. Francis Hotel. The program follows:

WEDNESDAY MORNING, OCTOBER 22

COURSE IN ELECTROCARDIOGRAPHY

Presiding: Arthur Selzer, M.D.

9:00-9:30—Electrocardiographic Differential Diagnosis of Acute Myocardial Infarction—Malcolm Watts, M.D.

9:30-10:00—The Effect of Exercise Upon the Electrocardiogram—Herbert N. Hultgren, M.D.

10:00-10:55—Electrocardiographic-Pathological Conference. Panel: Francis L. Chamberlain, M.D., David A. Rytand, M.D., Hans H. Hecht, M.D., Robert L. Smith, M.D., and Maurice Sokolow, M.D.

10:55-11:10—Intermission.

11:10-12:05—Common Errors in the Interpretation of the Electrocardiogram—Hans H. Hecht, M.D.

Wednesday Afternoon

HEART DISEASE IN CHILDHOOD

Presiding: John A. Anderson, M.D.

1:30-2:20—Uses of Digitalis, a Panel Discussion. Moderator Harold H. Rosenblum, M.D.
Participants: Hans H. Hecht, M.D., Margaret Maroney, M.D., Lt. (j.g.) Irving Fine (MC) USNR, and Saul J. Robinson, M.D.

2:20-2:40—Certain Aspects of Electrocardiography in Infants and Children, Margaret Maroney, M.D.

2:40-3:15—Cardiovascular and Renal Adjustments in Polycythemic Subjects, Hans H. Hecht, M.D.

3:15-3:30—Intermission.

3:30-5:00—The "Total Child" with Rheumatic Fever, a Round Table Discussion. Moderator: John A. Anderson, M.D. Participants: pediatrician, Bruce Johnston, M.D.; cardiologist, Harold H. Rosenblum, M.D.; psychiatrist, Hale F. Shirley, M.D.; psychologist, Mrs. Elizabeth Bing; nurse, Mrs. Frances Parchman; medical social worker, Mrs. Hazel Studdard; group worker, Miss Carol Young; dietitian, Miss Jean Zumwalt; occupational therapist, Miss Carolyn Zumwalt.

THURSDAY MORNING, OCTOBER 23

Presiding: Richard McLaughlin, M.D.

9:00-9:30—Overtreatment of Congestive Heart Failure—David A. Rytand, M.D.

9:30-10:10—The Physiological Basis for Oxygen Therapy—Julius H. Comroe, Jr., M.D.

10:10-10:20—Intermission.

10:20-10:35—Physiological Aspects of Pericardial Effusion—Victor Richards, M.D.

10:35-11:00—Treatment of Acute Tuberculous Pericarditis—Forrest M. Willett, M.D.
The Surgical Treatment of Constrictive Pericarditis—C. Sidney Burwell, M.D.

Thursday Afternoon

Presiding: John J. Sampson, M.D.

1:30-2:30—The Heart in Pregnancy—C. Sidney Burwell, M.D.

2:30-3:15—Symposium on the Heart in Pregnancy, Moderator: John J. Sampson, M.D. Participants: Ralph Benson, M.D., Ellen Brown, M.D., C. Sidney Burwell, M.D., Robert W. Churchill, M.D., Charles McLennan, M.D., and Ernest Page, M.D.

3:15-3:30—Intermission.

3:30-4:00—Selection of Candidates for Mitral Valvulotomy —Herbert N. Hultgren, M.D.

4:00-5:00—Discussion of the Treatment of Hypertension, a Symposium—Francis L. Chamberlain, M.D., William J. Fleming, M.D., Maurice Sokolow, M.D., and Clarence M. Tinsley, M.D.

Thursday Evening

Annual Dinner Meeting, 7:00 p.m.

Medical Problems in Korea—Frank Gerbode, M.D.

FRIDAY MORNING, OCTOBER 24

SYMPOSIUM ON PERIPHERAL VASCULAR DISEASE

Chairman: Alfred F. Goggio, M.D.

9:00-9:30 — Surgical Treatment of Peripheral Vascular Obliterative Lesions—Edwin Jack Wylie, M.D.

9:30-10:00 — The Prognosis and Treatment of Necrotic Pedal Lesions—Rutherford S. Gilfillan, M.D.

10:00-10:10—Intermission.

10:10-11:00—A General Discussion of the Sympathetic Nervous System from the Physiological and Pharmacological Point of View—Julius H. Comroe, Jr., M.D.

11:00-12:00—Thromboembolic Disease, a Panel Discussion. Moderator: Ellen Brown, M.D. Participants: Paul Aggeler, M.D., Roy Cohn, M.D., Frank Leeds, M.D., Benson R. Roe, M.D., John J. Sampson, M.D., and Arthur Selzer, M.D.

Friday Afternoon

1:30-5:00 (at Laguna Honda Hospital)—Presentation of Patients with Various Types of Heart Disease. Presiding: J. Marion Read, M.D. Participants: Henry Brainerd, M.D., C. Sidney Burwell, M.D., Julius H. Comroe, M.D., Maurice Eliaser, Jr., M.D., Roberta Fenlon, M.D., Marlow B. Harrison, M.D., Hans H. Hecht, M.D., Hilliard J. Katz, M.D., J. H. Lewis, M.D., Lester S. Lipsitch, M.D., Clayton D. Mote, M.D., Charles A. Noble, Jr., M.D., Ernest S. Rogers, M.D., Arthur Selzer, M.D., Norman Sweet, M.D., and Clarence M. Tinsley, M.D.

GENERAL

The sixth annual **conference and annual meeting of members** of the California Division of the American Cancer Society will be held on October 3 and 4 at the Clift Hotel in San Francisco. The program follows:

OCTOBER 3

10:00 a.m.—Registration and Opening Session.
Address by Dr. Michael Shimkin, director of the Laboratory of Experimental Oncology, University of California —"The Nature of Cancer and Its Effect on the Human Being."

12:15—Luncheon.
Address—"Where the Cancer Dollar Goes."

2:00—Workshop sessions on special Cancer Society programs: county organization; techniques of lay education; the lay service program; fund-raising and Federation.

7:00—Annual Dinner Meeting.
Address—Dr. Charles Lund, Boston, president of the national American Cancer Society—"Contributions from Recent Cancer Research to the Detection and Treatment of Cancer."

OCTOBER 4

10:00—Annual Meeting of Members.

The California Division of the American Cancer Society cordially invites all the members of the California Medical Association to attend any or all of the meetings.

POSTGRADUATE EDUCATION NOTICES

UNIVERSITY OF CALIFORNIA AT LOS ANGELES SCHOOL OF MEDICINE

Progress in Medicine — Annual Evening Medical Lecture Series—Room 121, Business Administration and Economics Building (BAE), Los Angeles Campus (Westwood).

Date: September 22-December 8, 1952, 7:30-9:30 p.m., Monday evenings.

Fee: $50.00.

PROGRAM

September 22—What Is New in Endocrinology?—Peter H. Forsham, M.D., associate professor of medicine and pediatrics, director of Metabolic Research Unit, University of California, San Francisco.

September 29—The Treatment of Collagen Diseases — Joseph E. Giansiracusa, M.D., assistant professor of medicine, University of California, San Francisco.

October 6—Indication for Bone Marrow Examinations—William S. Adams, M.D., assistant professor of medicine, University of California, Los Angeles.

October 13—Diagnostic and Therapeutic Management of the Jaundiced Patient—Theodore L. Althausen, M.D., professor and chairman, Department of Medicine, University of California, San Francisco.

October 20—Diagnosis and Management of Hemorrhagic Disorders—William N. Valentine, M.D., associate professor of medicine, University of California, Los Angeles.

October 27—Diagnosis and Management of Fungus Infections—Victor D. Newcomer, M.D., assistant clinical professor of medicine, University of California, Los Angeles.

November 3—Cirrhosis of the Liver and Chronic Hepatitis—Rubin L. Gold, M.D., assistant clinical professor of medicine, University of California, San Francisco.

November 10—Heart Disease in Small Infants and Children—Forrest H. Adams, M.D., associate professor of pediatrics, University of California, Los Angeles.

November 17—Pain: Its Significance as a Symptom of Disease—Augustus S. Rose, M.D., associate professor of neurology, University of California, Los Angeles.

November 24—Recent Advances in the Use of Antibiotic Agents—William L. Hewitt, M.D., associate professor of medicine, University of California, Los Angeles.

December 1—The Acute Abdomen—Orville F. Grimes, M.D., assistant professor of surgery, University of California, San Francisco.

December 8—Use and Abuse of Gynecological Endocrine Therapy — Irregular Vaginal Bleeding — Edmund W. Overstreet, M.D., associate professor of obstetrics and gynecology, University of California, San Francisco.

Contact: Application or requests for information concerning this course should be made to Thomas H. Sternberg, M.D., Head of Postgraduate Instruction, Medical Extension, University of California, Los Angeles 24.

UNIVERSITY OF CALIFORNIA SCHOOL OF MEDICINE

Medicine for General Practitioners—East Oakland Hospital, 2648 East Fourteenth Street, Oakland.

Date: September 23 through December 9, 1952. Tuesday Evening Lectures. Time: 8:00 p.m.

Fee: $50.00.

This course is approved by the California Academy of General Practice for 25 hours of postgraduate work.

Evening Lectures in Medicine—Mills Memorial Hospital, San Mateo.

Date: September 25 through December 11, 1952. Thursday Evening Lectures. Times: 8:00 to 10:00 p.m.

Fee: $50.00.

The first six lectures in this series have been prepared specifically for internists, and the balance of the lectures are intended for physicians who practice general medicine.

Contact: Stacy R. Mettier, M.D., Head of Postgraduate Instruction, Medical Extension, University of California Medical Center, San Francisco 22.

UNIVERSITY OF SOUTHERN CALIFORNIA SCHOOL OF MEDICINE

Recent Advances in Internal Medicine—Course 842—San Diego County Medical Library, Room 1410, Medico-Dental Building, 233 A Street, San Diego 1.

Date: September 17 through December 10, 1952, 8:00-10:00 p.m., Wednesday Evenings.

Fee: $50.00.

Recent Advances in Diagnosis and Treatment—Course 843—Kern General Hospital Auditorium, Bakersfield.

Date: September 18 through December 18, 1952, 8:00-10:00 p.m., Thursday Evenings.

Contact: Gordon E. Goodhart, M.D., University of Southern California Medical Extension, Box 158, 1200 North State Street, Los Angeles 33.

INFORMATION

New Minimum Wage Orders for Women and Minors

(Prepared by the State of California Division of Industrial Welfare)

CALIFORNIA'S new minimum wage of 75 cents an hour for women and minors went into effect on August 1, 1952.

The revised rate is contained in new minimum wage orders recently enacted by the Industrial Welfare Commission. These orders enforced by the Division of Industrial Welfare of the Department of Industrial Relations regulate minimum wages, maximum hours and working conditions for women and minors in all industries in California except agriculture and domestic service in private households.

The new regulations applicable to women employed in professional offices are contained in Industrial Welfare Commission Order 4-52 covering women employed in professional, technical, clerical and similar occupations.

Some of the most important provisions of this order which will affect women employed in these establishments are:

1. The minimum wage is increased to 75 cents an hour; provision is made for a 60-cent rate which may be paid to limited numbers of minors and learners.

2. Women working in bona fide executive positions will be exempt from jurisdiction of the order if they receive $350 or more per month.

3. Employment of women and minors will be limited to six days, of eight hours each, per week with exceptions for short-hour workers. Previously, weekly hours were limited to a maximum of 48 hours without limit on the number of days.

4. The minimum time that may elapse between the end of the employee's work day and the beginning of the next day will be eleven hours; also, in the event that the employee's day involves a split shift, it will be required that the eight hours of work must be performed within a period of thirteen hours.

5. It is permissible to work more than eight hours a day and 48 hours a week in emergencies, provided that time and a half the regular rate of pay be paid for such overtime.

6. Accurate time records must be kept showing all starting and stopping time which must be recorded when it occurs; also total daily hours must be shown. Meal periods must be recorded unless the firm has an established meal period during which operations cease. Total hours worked during the payroll period and total wages paid shall be kept on the same record.

7. Each employee is entitled to rest periods in the ratio of ten minutes for each four hours of working time or major fraction thereof.

8. The applicable order must be posted in the establishment of each employer.

The Division of Industrial Welfare is mailing copies of the revised orders to all firms on its mailing list. Employers who do not receive copies or who wish extra copies should contact the division office nearest them. Offices of the division are located in San Francisco, Los Angeles, Oakland, Sacramento, Fresno, Long Beach and San Diego.

Student Activities at University of California School of Medicine

THE SCHOOL YEAR 1951-52 marked the beginnings of two new student organizations at the University of California School of Medicine. The Associated Students of the School of Medicine, the official student organization, represents all the students. The Student American Medical Association is a new national medical student organization which has chapters at about forty medical schools.

One of the first projects of the Associated Students (ASSM) was to establish an orientation program. During the summer a letter of welcome was sent to the incoming freshmen. During registration week the freshmen were met by a group of students from each of the classes, who gave them expert advice on all phases of medical school, from the very practical to the philosophical: purchases of books and equipment; the art of survival in medical school; extracurricular activities; a student's, or worm's eye, viewpoint on school in general; and a student's welcome into the medical fraternity. Similar orientation has also been extended to the sophomore and junior classes by the classes above them.

With the aid of faculty advisors an investigation was begun of hospitalization insurance for medical students during the summer when they are not covered by the student health plan, and for their families during the entire year. A student committee investigated many policies but found none that was suited to the particular needs. SAMA delegates to the convention of the California Medical Association last April told the Association of efforts in this regard and requested aid in the solution of the problem. Dr. H. Gordon MacLean, past president of C.M.A., is now attempting to secure a suitable

policy. Along this same line, work was begun to establish a medical student emergency fund.

In the past, seniors have been unable to obtain adequate information about internships. In the belief that it would be of value to learn from interns their impressions of internship, a questionnaire was sent to all University of California School of Medicine graduates who were interning, as well as to a number of interns who had been graduated from other schools. A file of the replies, available to students, is expected to expand and to be of increasing value each year.

The Council of the ASSM was invited to meet with the steering committee of the faculty-student preceptorship program. Student opinions as to the successes and shortcomings of the program were requested. It was agreed that the ASSM should take some responsibility in promoting student activity in the preceptor program.

The SAMA held its second national convention in Chicago in December 1951. Delegates were present from 35 schools. The expenses of the California delegates were paid by the California Medical Association. Many items concerning medical students as a group were considered. The first issue of the *Journal* of the SAMA was released. This excellent journal, published nine times a year, contains articles of interest to students and to the profession in general; some are written by prominent men of medicine and some by medical students.

Two student delegates were invited to the C.M.A. convention in Los Angeles last April and there they learned much about the nature and workings of the C.M.A.

A number of other projects have been started: the interclass sale of used books, and plans for establishing book-sharing pools; a student lounge at San Francisco Hospital; a clarification of the hospital policy concerning the medical facilities available to students' families; the promotion of interest in the Alumni Association.

Of major concern was the problem of student-faculty relationships. At an informal meeting of all interested students and about a dozen members of the faculty, representative of all divisions of the school, there was general discussion of medical school problems from both the faculty and student viewpoints. It was discouraging, at times, to learn that present-day student problems are not too different from those existing in the student days of the faculty members. On the other hand, it was heartening that the faculty was cognizant of the problems and was already working toward solution of them. It was evident that such meetings, with free exchange of ideas, would be most effective in making medical school life more satisfying.

It was proposed by several members of the faculty at the meeting that students' suggestions regarding the teaching program would be of value to the curriculum committee and to the heads of divisions of the medical school. After the idea had been discussed with faculty advisors and with the division heads, a critique and evaluation was submitted.

FREDERICK J. SOBECK
Vice-President, SAMA
University of California School of Medicine
San Francisco

THE PHYSICIAN'S *Bookshelf*

MODERN ELECTROCARDIOGRAPHY — Volume I — The P-Q-R-S-T-U Complex. Eugene Lepeschkin, M.D., Assistant Professor of Experimental Medicine. The Williams and Wilkins Company, Baltimore, Md., 1951. 598 pages, $12.00.

In recent years so many new books on the electrocardiogram have appeared that another might appear to be repetitious and superfluous. Actually, this book is unique among modern text books on the electrocardiogram and will be of tremendous value to those who wish detailed and fundamental knowledge on this subject.

This first of two volumes covers the literature from 1934 until 1950 on the origin and form of the electrocardiogram and includes almost 10,000 references. The author has attempted to review and digest this tremendous body of information and at the same time inject his own views in controversial matters. In the opinion of the reviewer, he has been most successful in this attempt and has achieved his purpose of presenting a "comprehensive but concise summary of the progress of electrocardiography since 1933."

The nature of the book is such that it will be of greatest value to those who have some background of information and training in electrocardiography and who wish to gain further knowledge. As a reference text, it cannot be equalled by any modern publication in English, and should be included in the library of every investigator and practicing physician interested in the electrocardiogram.

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MEDICAL MILESTONES. Henry J. L. Marriott, M.D., The Williams and Wilkins Co., Baltimore, 1952. 293 pages. $3.50.

This is a readable single volume interpretation of the major medical advances of the past generation. It differs from much medical reporting in that it is neither too sensational nor hysterical. It calls attention to the drawbacks in some of the "miracles" as well as their accomplishments: for example, more time is spent in debunking the antihistaminics as "cold cures" than in telling of their full uses. There is a glossary of key technical words placed in attractive footnotes whenever such words are introduced.

The reviewer finds minor faults: Chapter five uses the proprietary term Chloromycetin throughout—without mention of the USP term chloramphenicol. In the table of contents terramycin is not mentioned although it is discussed equally with aureomycin in chapter seven. And although the author deplores "medical shrapnel" in the treatment of anemia, he condones it in the treatment of urinary infections.

On the whole, this book is a good, solid, well-balanced summary which the doctor may recommend to his patients and friends who desire to drink more deeply of the Pierian spring of medical knowledge. For that matter, the doctor himself may well read it to advantage!

RHEUMATIC DISEASES—Based on the Proceedings of the Seventh International Congress on Rheumatic Diseases. Prepared by the Committee on Publications of the American Rheumatism Association, Charles H. Slocumb, M.D., Chairman. W. B. Saunders Company, Philadelphia, 1952. 449 pages, $12.00.

The title "Rheumatic Diseases" is deceiving, for this volume is little more than the collected papers of the Seventh International Congress on Rheumatic Diseases, held in May, 1949. It is not a textbook of rheumatology, although advertisements (J.A.M.A. February 23, 1952) suggest that it would serve this purpose. The book is divided into chapters, but none of the sections is complete, although the discussion of fibrositis and psychogenic rheumatism is relatively lengthy. The section on the effect of cortisone and ACTH on rheumatoid arthritis and rheumatic fever by Hench et al is essentially the same material presented a month after the initial announcement of their work in April, 1949. Although reportedly brought up to date, there are only a few 1950 references in the bibliography and none after that year. However, there are authoritative papers on many phases of arthritis of importance primarily to rheumatologists, but also of interest to internists, physiatrists, orthopedists, pathologists and investigative workers in this field. The papers are too numerous to mention, but perusal of the table of contents would yield at least several articles of interest to most physicians.

There is a trend now toward publishing proceedings of meetings and freeing periodicals for more current publications. While this might be a worthwhile move, these collected papers presenting timely articles should not take three years to get into print if they are to retain much of their value. Further, they should be presented as proceedings or collected papers and not be confused with texts or well-rounded presentations of the material—a near impossibility with numerous contributors, despite careful editing.

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DISEASES OF THE NOSE, THROAT AND EAR—A Handbook for Students and Practitioners—Fifth Edition— I. Simson Hall, M.B., Ch.B., F.R.C.P.E., F.R.C.S.E., Surgeon to the Royal Infirmary, Edinburgh. The Williams and Wilkins Company, Baltimore, 1952. 463 pages, 8 colored plates, $4.00.

This volume is designed to meet the needs of the busy practitioner and the student.

The preface to the 5th edition written by the author takes up only half of a small page but is adequate. It informs one that this edition contains no radical change in size; but changes in treatment which have been proved and accepted and been introduced where appropriate, and that controversial matters have been avoided.

For those who are not acquainted with the preceding editions, this volume, although primarily set up for the medical student and the general practitioner, is a worthwhile addition to the otolaryngologist's library.

SURGICAL FORUM—Proceedings of the Forum Sessions 37th Clinical Congress of the American College of Surgeons, San Francisco, November, 1951. W. B. Saunders Company, Philadelphia, 1952. 667 pages, 290 figures. $10.00.

This is a book which represents the proceedings of the forum on fundamental surgical problems presented annually at the American College of Surgeons meetings. It, therefore, consists solely of reports on the experimental work that is being conducted the country over in the field of surgery.

It does not limit itself to any one surgical specialty, but covers research work and all surgical specialties. It is an excellent review of experimental work in the surgical centers of the United States. It will be valuable, therefore, to people interested in conducting research in their own hospitals. It is not of value to anyone wishing to acquaint himself with the latest developments in clinical surgery, except as these discussions can be transplanted on an experimental basis again to clinical problems.

It is recommended, therefore, to people interested in surgical research, but it is not the kind of book that the man who is interested in clinical surgery alone would profit by securing for his library. All of this work will appear sooner or later in one of the leading surgical journals. It reports the type of material that is seen in the leading surgical journals on experimental work but is about one year ahead of time.

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PRINCIPLES AND PRACTICE OF AVIATION MEDICINE—Third Edition—Harry G. Armstrong, M.D., F.A.C.P., The Surgeon General, United States Air Force. The Williams and Wilkins Company, Baltimore, 1952. 476 pages, $7.50.

This book is the standard text for flight surgeons for the armed forces, and as such the third edition has maintained its previous excellent qualities and over-all coverage of the field. There is no doubt that General Armstrong has wide experience in this field, and the text lives up to his impressive records.

It might be carping, but there are many suggestions for future editions as well as omissions which seriously detract from the value of this volume.

The order of the chapters leaves something to be desired. The chapters on Pilot Selection and Physical Examination of the Pilot precede the physiological problems of aviation medicine, and the chapter on Noxious Substances precedes the chapters on the Atmosphere, Altitude Sickness, and Oxygen in Aviation.

Numerous photographs of obsolete equipment which appeared in the previous edition are still maintained for little reason except, perhaps, for historical value.

A thorough chapter on respiratory physiology with more basic information and less application for equipment would be helpful. Similarly, the chapter on Speed and Acceleration elaborates on radial and rotary acceleration, at the expense of almost completely ignoring linear acceleration.

There is also no mention of the prone position as a possible method of ameliorating the effects of radial acceleration, and one-third of a page is devoted to the anti-blackout suit and one page to an obsolete crash helmet, while the newer work and developments by groups other than the Army are totally ignored. This exclusion of scientific work by groups other than the Army reaches almost embarrassing proportions when the excellent basic studies by the Mayo Clinic group on aeroembolism and aeroemphysema are barely mentioned. The basic endeavors of the Navy research groups on the anti-blackout suit and on linear acceleration and crash protection do not appear. The excellent helmet research by the group at the University of Southern California is nowhere to be found. The fine work on equilibrium by Graybiel with the U. S. Navy is omitted. The account of parachuting is antiquated, and no mention of Starnes' phenomenal free fall of 30,000 feet in 1943 is made. Most of this and other pertinent material is now declassified and should be contained in such an authoritative text for completeness.

Discussions of gravitational and anti-gravitational problems, ionosphere, space travel, jet and rocket aircraft and associated medical problems would be of current and future value to the student of aviation medicine. The bibliography at the end of most chapters persists in quoting work previous to World War II. The index is meager—but four pages for 460 pages of text replete with technical terms. In general, not much new material appears in the third edition that was not present in either the first or the second edition. However, this would not be a serious deficiency for any but the expert in aviation medicine.

It is evident that much information is withheld because of obvious military security which has not as yet been released. General Armstrong is faced with the difficult task of choosing the most significant data from a rapidly advancing field, and he has done an admirable job.

This book is recommended highly to the student entering upon aviation medicine for a complete and well-oriented view of the subject.

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DYNAMIC PSYCHIATRY — Transvestism — Desire for Crippled Women—Volume Two. Louis S. London, M.D., Corinthian Publication, Inc., 114 East 32nd Street, New York 16, 1952. 129 pages, $2.50.

The subtitle of this book is "Transvestism—Desire for Crippled Women." The case described is considered of importance because of the patient's psychopathological interest in crippled women, dwarfs, freaks, cross-eyed, bearded women, and because the patient sometimes masqueraded as either a male or female cripple. Transvestism is defined as "a form of sexual deviation in which the person desires to play the role of the opposite sex by crossed dressing." Following a chapter on the anthropological and historical references of the subject, 50 psychobiographic fantasies drawn by the patient and described in his own words are presented. The third and final chapter is concerned with the presentation of the case history; description of dreams related to shoe fetishism and transvestism, and at the conclusion a short description of the psychodynamics of the problem. The interpretation is obviously psychoanalytically oriented and emphasizes the importance of the Oedipus complex, latent homosexuality, the castration complex, sado-masochism, exhibitionism, shoe fetishism, and transvestism. This little volume is essentially a case report and will be of interest primarily to sexologists and psychoanalysts.

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STUDIES IN MEDICINE — A Volume of Papers in Honor of Robert Wood Keeton. Charles C. Thomas, Publisher, Springfield, Ill., 1951. 396 pages. $8.50.

This volume is a "Festschrift" prepared by colleagues of Dr. Robert Wood Keeton to celebrate his career in academic medicine, on the occasion of his retirement from the chair of medicine at the University of Illinois.

The chapters consist of unrelated topics by the faculty of the University of Illinois and Northwestern, with a few articles from other sources. The titles vary from "The Art of Using Lantern Slides" by Thomas S. Jones, to "The Microdetermination of Lipids — The Synthesis of Labile Phosphorus Compounds of the Phospholipids by Reaction with Monobasic Sodium Phosphate" by A. B. Kendrick.

Those readers who are interested in noting some of the investigative work being done in Chicago will find this volume of interest.